THESIS ON

PULMONARY PHTHISIS

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PULMONARY PHTHISIS.

This is defined by Coates as the term applied to "cases in which the lungs are effected by a progressive lesion, the ordinary and regular result of which is destruction of lung tissue and formation of cavities; cases in which this occurs from any other cause than tubercle are so few that Phthisis Pulmonalis may now be regarded as synonymous with local Tuberculosis of Lung."

This disease has long been one of the most disastrous to which the human race is subject and the more so that its ravages are made chiefly on the young. At the present day in spite of improved social circumstances and better hygienic surroundings, it bulks largely among the causes of death. (248) Ransome says that in 1838 it was answerable for 38 deaths per 10,000 inhabitants in England and Wales, but that this has been gradually reduced till in 1895 the death rate was only 14 per 10,000. (223) M'Fadyean says that one seventh of a population and from 6-20% of cattle suffer from its ravages. It is found all over the habitable globe, no special country or climate being altogether exempt from it. (142) Hirsch says of England that the lowest phthisical death/

death rate is found in the agricultural counties, the highest in the cities with many places of manufacture and trade.(143) Of France, he says, as in other countries it is the great centres of commerce, trade and manufacture that form the chief seats of the malady.(144) On the other hand he notes that among the Kirghiz of the Steppes it is unknown and also that it is much less frequently met with in high-lying(145) places than in those at lower elevation or sea level, e.g.,(146) in large towns of Central and South America; on the plateaux of the Andes it is rare, or almost unknown in spite of their being industrial and by no means models of sanitation.

In the present day owing to the causes being better understood, not only is the death rate much lower, but the course of the disease is longer and in a considerable number of cases complete or partial recovery takes place and the patient in enabled to live out a life of average duration and to follow some healthy and not too arduous avocation. Up to the middle of the 19th Century, Phthisis was looked on largely as an inherited constitutional disease, but about that time experiments began to be made on the possibility of communicating the disease by inoculation, which changed its standing and culminated/

culminated in the present view that it is largely an infective disease and may occur in those of vigorous constitution if exposed to infection and debilitating surroundings, while weakly persons with inherited predisposition escape if they live a healthy open air life.
(163)

Koch in his paper on Etiology of Tuberculosis gives Klenke in 1843, the credit of having first successfully produced extensive Tuberculosis of lungs and liver by inoculation with portion of (294) miliary and Tubercular infiltration from man. Villemin in 1865 corroborated this by his own experiments. Saunderson and Fox in 1868 in England and Cohnheim and others in Germany found that by inoculation of supposed non-tubercular material they produced tubercular lesions. (58) Cohnheim however, in repeating his experiments with greater precautions got negative results and was led to conclude that in his first results unintentional tubercular infection had occurred.

The next step in advance was made by Koch in his discovery of the Tubercle Bacillus as the active element in the propagation of disease. By staining methods specially developed for the purpose, he demonstrated the bacillus in recent grey tubercle from/

from an animal killed three or four weeks after inoculation.(164) By using an alkaline solution of methylene blue he detected fine rodlike forms, but owing to the difficulty of detecting them in sections he used a concentrated watery Solution of vesuvin to stain tissues brown,(165) while the Tubercle Bacilli were left blue.(165) He now recommends Ehrlich's method modified by Wiegert which stains Bacilli violet and a counter stain in sections of tissue of methylene should be used.

(295) Ziehl-Neelsen's which replaces the aniline used in Ehrlich's stain by 5% carbolic acid is also an excellent stain. A lens magnifying 500-800 diameters is necessary to distinguish the Bacilli.

(166) Bacillus Leprae behaves in a similar way to the Tubercle Bacillus with the above dyes, but may be distinguished by the fact that it stains with Wiegert's method for staining nuclei, which Tubercle Bacillus does not.(13) In urine the smegma bacillus may be mistaken for Tubercle Bacillus as it is acid fast also. The Tubercle Bacillus is a fine short rod occurring mostly in groups, - isolated rods show only ordinary molecular movement. In length they vary from $\frac{1}{4}$ - $\frac{1}{8}$ diameter of a red blood cell

(1.5 - 3.5 m.) - no joints are perceptible - their breadth is constant when the same method of staining is used, but when stained with methyl blue they appear thinner than when stained with methyl violet. The rods generally show bends and often a curve approaching first stage of cork screw. (224) Nocard, Roux and Klein observed in cultivation of human Tubercle Bacillus long threads with lateral branches. (168) Koch says they have the power of spore formation, which gives the appearance of small bright bodies at intervals in bacillus. (169) The spores are oval, 2-6 being present in a bacillus. (249) Crookshank classifies Tubercle Bacillus as aerobe or facultative anaerobe. No growth on gelatine spore formation present, non-motile found in tubercular material only. (14) Bulloch at Tubercular Congress in London, says there is no evidence of genuine endospores formed by members of the group of bacteria to which Tubercle bacillus belong. (225) The Bacillus appears to be surrounded by a membrane consisting largely of fatty acid and their close adhesion in cultivation points to an enveloping cementing substance. (170) They may be heaped together in thick masses or often only in small numbers and are most evident where the Tubercular process/

cess is spreading. They cannot be demonstrated in caseous material though found at the spreading edges. (171) Koch first tried to cultivate it with nutrient jelly of meat infusion and with peptone solidified by gelatine at ordinary temperature of a room, but met with no success, after a number of experiments he hit upon solidified blood serum as the nutrient medium and found 37°C. to be the most favourable temperature. The blood serum of different animals seemed to form an equally good medium. (172) On coagulated egg albumen they did not grow. (173) The flesh of different animals only slightly susceptible to Tubercle Bacillus, dogs, rats, etc., gave an equally suitable infusion for culture. He got no growth on vegetable media, such as boiled potato, but (250) Sir H. Beevor, Kanthack and others have grown Tubercle Bacillus on potato at ordinary temperatures. (226) Woodhead quotes experiments carried out by Roux, Nocard and H. Beevor which seem to show that Tubercle Bacillus can become used to a saprophytic existence growing luxuriantly, but losing virulence. (227) Also Coppen Jones as saying that Tubefcle Bacillus is simply a phase in the life history of a higher fungus and that its pathogenetic and parasitic existence/

tence are merely accidental and temporary - that at certain stages it is sepraphytic and as Sanders has observed grows more luxuriantly on vegetable than ordinary animal proteid media. (174) Koch, on the other hand, says that as the lower limits of temperature for growth of Tubercle Bacillus are not reached by summer heat and growth is very slow unless protected, it would be checked by more rapidly growing organisms before the life cycle was completed, therefore it is probably a true parasite only finding the conditions necessary for its existence in the animal or human organism, where it spores and passes through a complete cycle. Koch describes its growth on solidified blood serum as follows :- Great precaution must be taken in sterilizing all instruments used, as the Tubercle Bacillus being of slow growth are apt to be overgrown by any foreign bacteria which obtain a footing. (175) After planting crushed Tubercular tissue on a medium, no growth is seen till from 10 - 15 days, when whitish scales adhering to the surface of the serum are seen - probably separate scales develop from a single bacillus. when the scales are numerous they form a greyish white lustreless covering on the serum. If now, some/

some of these scales are spread out on a new medium, a membraniform colony results. It follows the course of the wire in inoculation, as the bacilli have no power of locomotion and only spread over by increase in mass of bacilli, which spread horizontally and when reaching fluid at the bottom of tube, they still spread horizontally over the surface. A characteristic of the Tubercle Bacillus is that it does not liquefy serum and spreads over the surface. Microscopical oberservation 5 - 6 days after inoculation shows the appearance of S shaped lines. These coalesce to form a membraniform appearance. The bacilli in these colonies are arranged with their long axis parallel to the long axis of the colony and are separated by a small space - probably due to cementing substance. The maximum development is generally attained in about 4 weeks and cultures carried on for 16 - 18 months show no perceptible change from the primary. (176) If fluid sterilized serum be used, a white film is formed on the surface and if seed sinks from shaking of tube, no growth takes place. The serum always remains clear. The most suitable temperature for growth of Tubercle Bacillus is 37° - 38°C. (177) At 42°C. no growth occurred and at 28° - 29°C. growth also ceases completely. Ransome in/

(251) in his experiments found that the organism was very tenacious of life under various conditions. (252) It resists both the action of the gastric juice and drying, and he found that it retained its virulence for 54 days in the dark in closed capsules. (253) He found, however, that the virulence of pure cultures is speedily destroyed by sunlight and fresh air. By exposing cultures to various degrees of light, damp, and fresh air by ventilation and in dwellings on different soils, he found that sunlight, fresh air, and dry sandy soil have a distinct influence in arresting the virulence of Tubercle Bacillus and that (254) in good surroundings the virulence was destroyed in less time than is necessary for pulverisation of sputum. (255) He also found that where there is sufficient organic matter in the air, either from impure ground air in damp cellars and ground floors, or from reek of human bodies in ill ventilated places, that Tubercle Bacillus can retain its life and virulence (256) at the ordinary temperature of a living room.

(15) Harold Coates collected dust from houses inhabited by consumptives, from floors, skirting boards, walls, shelves, etc., and by injection into guinea pigs tested its virulence and found in 66% of dirty houses the presence of virulent Tubercle Bacillus was proved /

ed, while in 50% of clean houses, it was present in dust. Samples taken from a general waiting room of railway station also proved infective. The waiting rooms of two large hospitals gave negative results. (16) Mitchell and Crouch at Denver at an altitude of 5,290 feet found that Tubercle Bacillus as expectorated on sandy soil is still virulent after 35 hours exposure to the direct rays of the sun, but after that time, the virulence is gradually diminished and lost. (228) M'Fadyen quotes Forster as saying that Tubercle Bacillus was killed in four hours by temperature of 55°C. and in one minute by 95°C. The value of fresh air, sunlight and dry sandy soil in arresting the growth and virulence of Tubercle Bacillus is amply demonstrated by the above facts.

The next step was to prove that Tubercular material or pure cultures of Tubercle Bacillus if inoculated into an animal would produce true tubercular lesions. (178) Koch in his inoculation experiments got the same results from both with the exception that the disease occurred more speadily when the pure culture was inoculated, as in the case of Tubercular material the Tubercle Bacillus had to be free/

free from the tissue in which it was embedded before it could act on its new host. He found that it could be introduced into the animal organism in three ways,

1. By inoculation into various parts of the body; subcutaneously into the Peritoneum, veins, anterior chamber of eye, etc.

2. By inhalation - here infection took place by the Respiratory tract - the lungs, pleura and bronchial glands being affected.

3. By feeding - the tonsils might be affected in this manner, or the intestine, the lesion beginning in the solitary glands and Peyer's patches and secondarily affecting the peritoneum and mesenteric glands and other structures.

(59)
Cohnheim had shown the extreme suitability of the anterior chamber of the eye as a site for observing the earliest results of inoculation. By that means he observed between the 20th and 30th day a number of small transparent greyish nodules on the iris, which itself becomes reddened and blurred. These multiply and extend. Panophthalmitis sets in followed by tuberculosis of neighbouring lymphatic glands, lungs, liver and spleen.

Koch/

(179)
Koch inoculated various parts of the animal organism - abdominal wall, peritoneum and anterior chamber of eye. The wound healed next day and for a fortnight there was no local result; then the nearest lymphatic glands enlarged and an indurated nodule appeared in the wound and shortly broke down and ulcerated. The animal then began to lose flesh and look ill and became breathless and died or was killed in four to eight weeks, when the following Post Mortem changes were noticed. Tubercular ulceration at point of inoculation; tubercular changes in neighbouring lymphatic glands and also in lungs, liver and spleen and by the microscope the characteristic structure of Tubercle and Tubercle Bacilli were demonstrated. He found that the
(180)
results varied with the dosage of Tubercle. In the case of the anterior chamber of the eye, with a large dose, in a few days intense iritis sets in and panophthalmitis and a large number of Tubercles were found in lungs, liver and spleen almost as soon as by venous infection, while the lymphatic glands
(181)
seemed to be passed over as if apparently they were flooded by such a large number of bacilli that many
(182)
break through and only a few are held back. With a small dose, a slow creeping spread of Tubercles over/

over the iris took place; the lymphatic glands at the angle of the jaw below the ear became enlarged and caseous, and later other organs were affected. (183) In the case of the peritoneum also, a distinct difference was noted according to the dose injected - if large, the omentum was much indurated and thickened and caseous in the centre and numerous Tubercle Bacilli were found in enlarged liver, spleen and peritoneum, but death occurs before tubercles are formed. (184) With a small dose, a disseminated tubercular eruption in peritoneum took place with nodules also in liver, spleen and omentum. (185) Injection of a pure culture into the veins caused an extensive and pretty equally distributed eruption of miliary tubercles in a very short time. These inoculation experiments show that in a definite time after the introduction of Tubercle Bacilli, a characteristic lesion results which tends more or less slowly to spread through the body following definite paths, the speed and intensity of result varying with the number of Tubercle Bacilli introduced.

INFECTION BY INHALATION.

In inhalation experiments the possibility of spontaneous tuberculosis has to be avoided by keeping/

in the animals experimented on away from possible communication with tubercular animals.(187) Spontaneous inhalation Tuberculosis may also be distinguished from artificial by the character of the lesion which in the former consists of a few large tubercular centres in the lungs with caseation and associated with this, enlarged cheesy bronchial glands,(188) while in the artificial form a large number of small tubercles are found in the lungs, due to the much larger number of germs inhaled.(186) By spraying with a pure culture of Tubercle Bacilli rubbed up with distilled water and diluted till almost clear, into a box inhabited by animals, it was found that in ten days dyspnoea appeared in some of them, a few died in fourteen to twenty-five days and on the rest being killed after twenty-eight days, a Post Mortem examination showed numerous tubercles in the lungs varying in size with the time the animal had lived. In those which lived longest, tubercles were also found in the liver and spleen. When the tubercles in the lung reached a certain size, extension by way of the alveoli could be recognised and chiefly embraced the centre of the lobule.

INFECTION BY ALIMENTARY CANAL.

With regard to feeding animals with tubercular material,(65) it was found that in from 12, - 21 days a local/

local lesion was present in the small intestine or caecum. It spreads thence to mesenteric and caecal glands, coeliac glands, liver, spleen, posterior mediastinal glands, bronchial glands and lungs.(66) In pigs, where the tonsil is very susceptible to tuberculosis, ulceration of tonsils with enlargement of cervical glands is frequently found in addition to the above. Where the tubercular material is not very virulent, the disease spreads slowly from intestine and mesenteric glands, a considerable time elapsing before the other organs are infected.(67) An important point brought out in the case of a calf, was that in one case there was no discoverable lesion in the intestine yet the mesenteric glands were infected and from a focus such as this,(68) a distant part of the body, e.g., the epididymis (in case of one pig) was found to be affected, infection probably taking place by way of the blood stream.(69) Even where a virulent material is used for feeding, a local lesion is always present, but may be small and undergoing calcification, while disease in the rest of the body is extensive and active.

In his inoculation experiments Koch found(190) that he got the same results in guinea pigs and rabbits, whether/

whether the tubercular material was derived from cattle, swine or human subjects and at that time considered the disease to be the same in all, only modified by the different soil.(191) Avian Tuberculosis he also looked on as being the same disease modified by the soil in which it grew.(192)(70) The bacillus of this latter , according to Clifford Allbut, grows at 43°C., which the human variety will not, and although inoculation of a guinea pig(71) only produces a slight lesion at first it is found that by transmission from guinea-pig to guinea-pig, a typical spreading lesion is at last produced.(17) Koch now however, disputes the identity of Tuberculosis in cattle and man, on the strength of experiments made within the last two years. He inoculated a number of young cattle, proved free of Tubercle by Tuberculin test, with pure cultures of Tubercle Bacilli taken from human Tuberculosis and in some cases sputum of consumptive patients direct. Some were fed with it, others inhaled large quantities in form of spray . None of them showed any symptoms and when killed six to eight months afterwards, no sign of tuberculosis was found. When cattle were inoculated with bovine tuberculosis on the other hand, after/

after a week's incubation severe tuberculous disorders of internal organs broke out in all infected animals. He got the same result with swine, sheep and asses. Opposed to this is a feeding experiment described in C. Allbutt. (72) Two calves were fed out of the same tub with 440 c.c. of sputum from two cases of Pulmonary Tuberculosis containing numerous Tubercle Bacilli. One killed in fifty-six days showed thirteen nodules of Tubercle scattered through Peyer's patches and also Tuberculosis of mesenteric glands, but none elsewhere. The second killed after 138 days showed no tuberculosis whatever. The first calf was a Jersey, a breed more subject to Tuberculosis than that to which the second calf belonged, viz., Shorthorn halfbred.

The above experiments demonstrate the three main avenues of entrance of the Tubercle Bacilli into the living organism. Of these, clinical experience shows that the commonest are by:-

I. Inhalation - infection taking place by the air passages.

II. By feeding - infection taking place by the alimentary canal.

III. By inoculation - infection occurring through the skin is not so common and is generally localised at the point of infection or nearest lymphatic glands.

(193)
Koch thinks that Tuberculosis of superficial glands is due to entry of Bacillus by scratches or breach of surface caused by skin eruptions. (257) A fourth method which is recognised as having occurred, but only as extreme rarity, is transmission of Tuberculosis to foetus through seminal fluid or mother's system. (18) C. Allbut says that in early life the bowel is the chief seat of infection. In youth the bowels may still be affected and the pharynx , nose, ear and cervical glands are liable to suffer, and by other means the bones. In later life, the lungs are chiefly affected, probably due to direct inhalation.

(194)
When Tubercle Bacilli are inhaled, if the ciliated epithelium of Trachea and Bronchial tubes be healthy they may be carried upwards to the outer air again without getting a foothold, but if there happens to be a spot denuded of this, such as may be found after measles, whooping cough, or frequent catarrh, or if lungs be crippled by old pleural adhesions, malformations of Thorax, or anything interfering with their free expansion or the free circulation through them or hindering the escape of secretions/

secretions they have a chance of establishing themselves and having once gained a footing, they spread to mediastinal and bronchial glands and lung itself. (227a) According to Woodhead only rarely does the lung precede the gland infection.

Where the tubercular material is introduced along with the food, Tuberculosis of the abdominal organs follows, or it may be of the tonsil with secondary infection of the cervical glands and perhaps extension to glands of upper part of Thorax and Pleura. This mode of infection is chiefly found in young children probably owing to the large amount of uncooked milk which enters into their diet. (19a) Koch however, asserts that it is extremely rare to find primary abdominal Tuberculosis even in children and that in the large majority of cases which he has examined post mortem, he has found evidence of previous focus in lungs. (19b) His evidence is, however, contradicted by English Hospital reports. The sources from which infection are derived are:-

1. The discharges of human consumptives and of these, by far the most important is the sputum, which if it be not destroyed by burning or some mode of disinfection, as soon as expectorated, may dry on sheets/

sheets or handkerchiefs, rags, etc., used to receive it and fall to the floor mingling with the dust, and if the surroundings are favourable may retain its vitality for long and being wafted about by currents of air, be inhaled by those who pass some time in sickroom. (18) Clifford Allbut quotes Flugge as saying that spray of spittle is even more dangerous than dry sputum. In cases with intestinal disease the excreta from the bowels also contains a definite quantity of Tubercle Bacilli and may form a source of infection if not carefully destroyed.

2. The other great source of infection is milk and meat from Tuberculous animals. (20) Koch has said that if this ever happens, it is a very rare circumstance, but in the meantime it is still subjudice and it is only wise to exclude every possible source, especially when clinical experience seems to point to the probability of it. (229) Tubercle Bacilli may also be transmitted in butter and cheese made from infected milk. (230) Tuberculosis is most commonly met with in cattle, swine and poultry - sheep very rarely. In case of cattle, the disease is much more common among cows than oxen/

oxen.(231) The statistics of slaughter houses in Munich, Berlin and Leipsic, on slaughtered cattle, give 2%, 4% and for several years 11% - 20% (Leipsic,)(232) but among cows alone 60 - 70% in a given district may be found tuberculous.(233) As regards milk, the Royal Commission concluded after a series of experiments, that Tuberculosis of the udder was necessary to render the milk infective.(21) Dr Lydia Rabinowitsh said she had found Tubercle Bacilli in milk of cows with sound udders.(22) Ravenal (Philadelphia) inoculated guinea-pigs with mixed milk from five Tubercular cows with sound udders and found 12.5% died of Tuberculosis.(23) Bang, Copenhagen, said that in his experience it was rare to find Tubercle Bacilli in milk when no tubercular lesions of udder were present. Out of 63 cows with advanced tuberculosis, but with healthy udders only 9 gave milk which produced tubercular lesions in guinea-pigs and only one which caused death of guinea-pig. The carcases of slaughtered animals are found chiefly to have the disease situated in the organs, lungs, lymph glands and serous membranes; very rarely in muscle and muscle juice, and as these parts are not eaten, but are removed in the dressing of the carcase, if proper precautions in handling and cleanliness of hands/

hands and knives be taken, a large part of the carcase of Tubercular animals, if muscle is in good condition, may be safely used. The safety may be rendered absolute by thorough cooking - ordinary cooking will destroy bacilli on surface, but meat must be thoroughly cooked through to destroy any that may be in centre of joint or of a roll of meat.

Effect of Tubercle Bacilli on Tissues.

(44)
Coates says the typical lesion in tuberculosis is the so-called miliary tubercle. (45) Baumgarten and Hamilton say that bodies having similar structure to miliary tubercle may be produced by small foreign bodies, but in these cases the lesion is non-infective. Before the discovery of the Tubercle Bacilli various theories had been advanced about these bodies. (1) Addison thought they were something more than a mere inflammatory process. (263) Rindfleisch thought they were due to a specific irritant applied to an endothelial surface. e.g., a lymphatic vessel, their frequent occurrence in the adventitia of capillary arterioles being due to the presence of lymphatics there. (60) Cohnheim quotes Klels as suggesting that they were of the nature of infective tumours/

tumours.(208)(209) Niemeyer held that they were secondary to a cheesy deposit in lungs or elsewhere.(87) Fagge quotes Virchow as describing them as lymphomata, while he himself suggests that they were the result(88) of a circumscribed irritation of epithelium or connective tissue.(264) Rindfleish describes three varieties of cells entering into their composition,

1. Large polygonal cells derived from the action of the specific irritant on the endothelial cell and from these he thought the other cells were derived.
2. The small round cells being produced by endogenous fission of nucleus and cell
3. while Giant cells were formed by endogenous fission of nucleus of large cell with failure of cell material to divide.

Cohnheim(61) thought the leucocytes were emigrated colourless blood corpuscles, but thought there was no proof of the origin of the component cells of Tubercle. Fagge quotes Hamilton as saying that lymphoid and epithelioid cells arise from connective tissue nuclei and the giant cell is formed from a single large connective tissue element. He also quotes Klein and Arnold as holding that the giant cell is formed by the fusion of a large number of connective tissue elements with persistence of nuclei.(296) Williams thinks that the irritation of

Tubercle/

Tubercle Bacilli produces proliferation of the fixed tissue cells whence are formed the epithelioid and giant cells. (196) Koch thinks that probably a wandering cell bearing a bacillus is transformed into an epithelioid and later a giant cell. (73) Metchnikoff holds that the epithelioid and giant cells are derived from mononuclear leucocytes and cells derived from vascular endothelium. (297) Baumgarten described the process minutely by observing changes that occurred after inoculation of Tubercle Bacilli into anterior chamber of eye of rabbit. He says that first the foreign body (Tubercle Bacilli) is encapsuled by granulation tissue, then an increase in the number of Tubercle Bacilli takes place, which force their way through the granulation tissue, and lying free in the intercellular spaces and fixed cells, by their irritation, (46) produce exudation of leucocytes and proliferation of fixed tissue cells, which form epithelioid and giant cells. In the giant cell, nucleus division goes on, but cell division does not take place. Owing to the irritation, an exudation of leucocytes takes place from the blood vessels, at first outside large celled tubercle, but penetrating into it and may transform it into a small/

small celled or lymphoid tubercle. If tubercle Bacilli are abundant and vigorous or accompanied by foreign microbes, there may be great production of leucocytes and the tubercles rapidly pass into the small celled condition. When the Tubercle Bacilli are few, and the cultivation is pure, the large celled non-inflammatory tubercle is most typically seen.

(195)
Koch says if an extensive portion of lung be involved by the inhalation of a quantity of the contents of a cavity rich in Tubercle Bacilli, no formation of separate tubercular nodules takes place, but we get tubercular infiltration, showing by lobar and lobular distribution that it starts from the Bronchi. The cellular elements in a miliary tubercle are generally grouped with the giant cell in or near the centre, surrounded by epithelioid cells and small round cells outside, getting lost in the surrounding tissue. (298) The Tubercle Bacilli, according to Williams are found chiefly in the epithelioid and giant cells, in the latter there seems to be a direct antagonism between the Tubercle Bacilli and Nuclei. (197) If the nuclei are at one pole, the Tubercle Bacilli are at the other; if there/

there are nuclei at both poles, the Tubercle Bacilli are equatorial. (234) Woodhead says that with the exception of giant cells, the Tubercle Bacilli in the human subject are extracellular, but that in cases tending to recovery they are found in the epithelioid cells. Owing to either a specific action of the Tubercle Bacilli or to shutting off of blood supply, the cells in the centre of the Tubercle caseate and lose all signs of any special structure, showing only a granular mass which contains few or no Tubercle Bacilli detectible by staining, probably owing to their passing into a spore bearing stage as the mass is still infective. From this original Tubercular focus, the disease spreads in four ways,

1. By direct continuity,
2. by lymphatic channels,
3. by blood vessels,
4. in case of lungs, by re-inhalation of tubercular expectoration into a healthy part of lung.

The lymphatic spread is shown by the distribution of miliary Tubercles in the neighbourhood of a tubercular focus or cavity and by affection of lymphatic glands and serous membranes. Where a miliary tubercle or tubercular focus opens into an artery or vein and both have been observed, the whole/

whole system is flooded and general miliary Tuberculosis results.

(258)
Mixed Infection. Along with Tubercle Bacilli other organisms are sometimes found in sputum, cavities and foci of disease in lungs, and in a case quoted by Koch (190), micrococci were found blocking capillaries and forming emboli in lung and spleen. (378) Jakowski and others also assert that streptococci are found in the blood of patients with hectic fever. Cornet found that rabbits inoculated with tubercular sputum, more especially from acute cases frequently die from septic processes in a few days. (259) The organisms found are pneumococci, Friedlander's Bacillus, Staphylococci, Streptococci, Micrococcus, Tetragonus and Bacillus Pyogenea Fetidus. They appear in cases where the soil is suitable, and resistance of patient impaired and his powers sapped by the tubercular process, to invade the part damaged by the tubercular process and by their activity, aid in breaking down the damaged tissue and promoting cavity formation. They will thus favour the spread of Tubercle Bacilli and may cause acute exacerbations in chronic or latent cases. (260) Absorption of their products would also seem to play a considerable/

considerable part in the production of hectic fever, loss of flesh, and sweating.

MORBID ANATOMY OF LUNGS IN PHTHISIS.

These present a very varied picture - the affected lung may be packed with discrete miliary tubercle associated with acute vesicular emphysema, or it may show catarrhal pneumonia with or without miliary Tubercles - or large tracts of caseous infiltration with ragged cavities, or Croupous Pneumonia with miliary Tubercles. In chronic cases it may be the seat of extensive fibroid changes showing cavities lined with fibroid material obliteration of alveoli by hypertrophy of interlobular septa, thickened pleura and associated with these emphysematous changes and dilatation of Bronchi. (90) It is rare to find the changes confined to one lung at death, by that time the second lung is almost always affected to some extent. The variety of change has given rise to much discussion as to whether Phthisis was due to the same cause in every case. (210) Laennee insisted that all cases were due to a specific new growth giving rise in some cases to miliary tubercles, in others to tubercular infiltration and that cavity formation was due to the/

the growth, caseation and expulsion of the new growth. (2) Addison on the other hand held that the great cause of the lung destruction in Phthisis was inflammation. (211) Niemeyer held that chronic catarrhal pneumonia was the main element in the vast majority of cases and that tubercles were a complication of this with a casual connection. (212) Cohnheim held that if a catarrhal pneumonia caseated, this was evidence of its tubercular nature from the outset. (62) Fagge holds to the identity of all cases of Phthisis and Koch's discovery of Tubercle Bacilli has decided the case in favour of one cause being at the bottom of all, however much the further developement may be modified by complicating causes and circumstances . The great variety of appearances in the lung suggest that different modifying causes are at work in different cases. (91) Miliary Tubercle and tubercular infiltration may be put down as the direct result of the Tubercle Bacilli. Evidence of the action of other organisms, Pneumococci and Strepococci, is found in the pneumonic appearances so frequently found in Phthisical lungs, though their invasion may as Whittaker says, be secondary. (379) Fibroid changes seem to be of the nature of reparative changes/

changes induced by nature when the bacillus has been overcome by the organism. (299) Fibroid changes seldom or never show the tubercle Bacilli and are of a stable nature, not tending to break down and caseate as so many new growths in connection with Tubercle do.

The different changes in Phthisical Lungs may be classified as follows:-

Changes due to Tubercle Bacilli primarily with, in some cases, other organisms grafted on.	Miliary Tubercle Tubercular Infiltration Chronic Catarrhal Pneumonia Croupous Pneumonia Bronchitis Pleurisy Enlargement of Bronchial Glands. Vomicae.
Conservative Processes	Cretification Fibrosis
Secondary Results of Tubercular change	Emphysema Bronchial Dilatation.

(303)
Miliary Tubercles, are described by Williams as being of three varieties, Grey, White and Yellow. (92) Fagge classifies as Soft Grey, Yellow and Hard Grey. The difference between the two classifications consists in the fact that Fagge evidently does not consider the difference between white and yellow of sufficient importance to merit a special class for the/

the former, while he gives a special class to the hard grey, which Williams describes as a modification of the soft grey occuring when the disease becomes arrested and fibroid changes set in, giving the soft grey a hard semi-cartilaginous consistence. (304)

Williams describes the grey as being of the size of a millet to hemp seed - soft semi-transparent (305) often occurring in clusters like bunches of grapes, and with a tendency to caseate when they become transformed into the yellow, which are the size of a pinhead to a pea, soft granular amorphous and sometimes surrounded by a circle of pearly transparent material. They are generally associated with the grey and frequently occur in recemose groups with grey granulations leading from them where however, the disease becomes arrested, grey granulations become drier, harded and cartilaginous in consistence. (306) White granulations differ from the grey only in being softer and more opaque and containing a larger proportion of epithelioid elements. Miliary Tubercles appear to be very frequently of secondary origin and to arise from some of the tubercular poison of a pre-existing cheesy focus getting into the blood stream or lymphatics and/

and then setting up general or local infection. (213) (214) Niemeyer and Buhl lay stress on a pre-existing cheesy focus being present in some part of the body, not necessarily the lung, in nearly all cases of Miliary Tuberculosis. (93) Fagge, criticising the fact that in 30 out of 300 cases Buhl failed to find a pre-existing focus, suggested that a recent patch of Tubercles so situated as to grow into the Pulmonary Vein might cause general Tuberculosis. (200) Koch quotes a case of this kind where the microscope showed that Tubercle Bacilli had insinuated themselves into the blood vessels and caused a generalised eruption of grey granulations. Where blood infection occurs, the discrete grey tubercles are found in the various organs, lungs, liver and spleen in large numbers and within a short time of infection (2-3 weeks in Koch's rabbits). (94) Fagge remarks that the Tubercles are more abundant, larger and better developed in the upper than the lower lobes, even when distributed by the blood stream and explains this by proclivity of tissue playing a determinary part. (74) Percy Kidd corroborates this, saying that in Miliary Tubercle, the apical lesions are more advanced, indicating a special vulnerability of/

of this part. In other cases the eruption of miliary tubercles occurs only in the neighbourhood of a previously affected area and Niemeyer (214a) suggests that this limitation speaks to lymphatic infection. When tubercles are thus limited in distribution, the larger tubercles near the infecting focus are larger, yellow and caseating, while the more recent at the periphery, are grey and have not begun to caseate.

The Tubercles, (3) Addison says, are situated in the delicate filamentous tissue which forms the filmy parietes of the air cells. (270) Rindfleisch says they are found in the connective tissue of the lung, in the interalveolar tissue or interstitial connective tissue or pleura and sometimes in the Bronchial mucous membrane. (307) They occur singly or in groups of various size, aggregated to form compound tubercles (308) and in some cases forming large masses occupying several adjacent lobules or the greater part or one lung. (309) They either soften and caseate, in some cases breaking down to form cavities (310) or in favourable cases, become hard and cartilaginous and transformed into fibroid tissue. As a result of the inhalation of a large number of Tubercle Bacilli into the lung a condition is produced termed <u>Tubercular Infiltration</u>/

Infiltration. There may be no miliary tubercle in evidence or only at outlying parts where it is the evidence of secondary lymphatic extension. (311) A large section of lung is involved; this is filled with a yellowish infiltration caused by

1. Aggregation of compound yellow granulations packed so closely together as to lose their typical appearance.

2. Rapid caseation of inflammatory exudation.

This caseous pneumonia, as it is also called, readily breaks down in places to form cavities with irregular crumbling walls, showing no limiting membrane.

In addition to the above more purely tubercular changes, are to be found lesions of an inflammatory character, viz., Acute and Chronic Catarrhal and Croupous Pneumonia.

These Pneumonic changes form a very important element in the course of Phthisis. (4) Addison says that they constitute the principal source of lung destruction and excavation in Phthisis. They are found principally in the upper lobes at first, afterwards extending downwards, though exceptionally they commence at the base. The most frequent lesion is Catarrhal Pneumonia, though croupous changes/

changes are occasionally evident. (215) Niemeyer says every form of Pneumonia may, under certain conditions terminate in caseation, though the Chronic Catarrhal form far most readily does so, and it is therefore, the chief inflammatory lesion in Phthisis. It is secondary to a catarrh of the bronchial tubes and Rindfleisch (266) describes its onset as due to plugging of these tubes from swelling of their walls and accumulation of secretion in the lumen. Consequent on this atelectasis or collapse of alveoli takes place and in this affected part, secondary changes ensue; the blood vessels become gorged and oedema takes place followed by inflammatory changes which result in accumulation of catarrhal cells derived from alveolar epithelium. These cells then fattydegenerate and caseate. (267) This condition is sometimes only lobular, limited to the circumference of smaller and smallest bronchi and of such small extent that the lesion has sometimes been mistaken for miliary tubercle. (268) In other cases it may be lobar by more abrupt extension of inflammation from bronchi to the whole portion of the parenchyma which they supply.

More/

More rarely the exudation is of a croupous nature. Addison says that both red and grey hepatisation are found in phthisis, but are very apt to soften and break down. (312) Williams says red hepatisation is frequently found associated with miliary tubercle more commonly in lower than upper lobes. (47) Coates says the exudation into the alveoli is sometimes more like that of an acute inflammation consisting to some extent of round cells, and there are cases in which fibrin is present. (95) Fagge holds that true lobar pneumonia is an accidental complication of phthisis and Whittaker says that it is not identical with true Croupous Pneumonia. (75) Percy Kidd out of some thousands of necropsies, only found one undoubted case in which progressive tuberculosis of lungs was complicated with acute fibrinous pneumonia. Owing to the pressure of the accumulated exudations with consequent cutting off of blood supply and action of Tubercle Bacilli, caseation takes place and in unfavourable cases, softening of caseous mass occurs and ulceration through the bronchial wall followed by evacuation of the caseous area and consequent loss of lung tissue. (5) Addison says there is usually some attempt at repair leading to iron grey induration/

induration and granular induration- formation of fibrous tissue - with contraction of deposit and included lung tissue. (6) These attempts are, however, owing to defective nutrition, imperfect and sooner or later softening and excavation takes place . (269) Rindfleisch says that attempts at repair invariably rise from the interlobular connective tissue and pleurae, and exhibit the general characteristics of reactive inflammation at the periphery of the lobule.

Bronchial Changes.

The bronchi are the site of the earliest attack of the Tubercle Bacilli. (313) Williams says that the bronchi are implicated in all cases of phthisis and that in many, catarrh is the principal lesion. (314) He quotes Rindfleisch as saying that the first change in phthisis is tubercular infiltration of all the angles and projections of the terminal bronchi where they become continuous with the alveoli; well marked greyish nodules of tubercle form on these projecting surfaces and afterwards caseate. Later on, tubercles form a more or less complete ring round certain bronchi. The tubercle lies in the sub-epithelial layer and is well supplied with giant/

giant cells. The peribronchial tissue is infiltrated and the bronchus reduced in calibre.(48) Coates says the initial change consists in swelling of the mucous membrane of the bronchial wall and infiltration with leucocytes; the lining epithelium desquamates and along with accumulation rich in round cells blocks the tube.(49) A similar cell growth takes place in the peribronchial sheath. There is an extension to the alveoli which are occupied by large catarrhal cells derived from the alveolar epithelium.(51) In fibroid phthisis the bronchus contains round cells and desquamated epithelium; the wall is infiltrated with round cells and tubercles are present in the wall and surrounding connective tissue, but the lung alveoli are not affected to the same extent and the disease seems to advance rather by lymphatic infection to connective tissue than by direct extension to alveoli.(51) In the midst of the small celled infiltration of the bronchial wall and connective tissue, tubercles are found perhaps obscured by inflammatory infiltration, but showing giant cells.

Percy Kidd says the tuberculous growth, whether in bronchiole or air sac, at first consists of epithelioid cells, but in some cases the nodule consists/

sists of small round cells. Subsequently, large multinucleated cells, giant cells appear singly here and there. (216) In one form of phthisis Niemeyer says the development of tubercles seems to start in the mucous membrane of the bronchi. In the trachea and larger bronchi, granular patches, consisting of innumerable miliary tubercles or ulcers are found, and in the smaller bronchi, white and yellow tubercles are found alongside of purulent catarrh. (270) (271) (272) Rindfleisch says that associated with Broncho-pneumonia and also in the neighbourhood of cavities, tubercles are found in the mucous and submucous tissue of the bronchi.

Coates (52) says that Tuberculosis of the bronchi is frequent in connection with cavities, the mucous membrane becoming the seat of tuberculous ulcers, visible as more or less rounded erosions, with distinct white tubercles at their borders; the result of infection by cavity contents. (315)

Pleura:

There is generally inflammation of this membrane in the neighbourhood of tubercle. In chronic cases it frequently shows adhesions between the costal and parietal surfaces. These may be only over a limited surface, or it may be much thickened and adherent from base to apex with intercommunication of blood vessels. (316) The process is conservative, preventing Acute pleurisy

and/

and Pneumothorax. In acute cases of phthisis where the affected lung tissue is near the surface and there is cavitation, perforation may occur with resulting pneumo-thorax. (96) Serous and purulent effusions are also occasionally found. The Pleura is also sometimes in local patches showing infection from underlying caseous patch, or in acute tuberculosis ther may be extensive eruption of Tubercles.

The Bronchial Glands.

(317) They generally become enlarged and caseous and may be much affected, while the lung substance is almost entirely free. Indeed, Woodhead says they are always affected before the lung itself. (318) They may cause suffocation by pressure on Trachea but more frequently ulcerate through and leave a cavity. (53) In some cases they cretify. In fibroid form, the glands become fibroid and deeply pigmented.

Vomicae.

These are the result of the caseation and softening of infiltrated lung substance, which after a time ulcerates through the wall of a bronchi-

al/

al tube, thus making a communication with the external air and allowing the softened caseated material to be expectorated when a cavity is formed. (54) Coates says softening of the caseated mass begins in the situation of a bronchus. Cavities vary greatly in appearance. (202) In acute cases the wall shows plainly the alveolar structure filled with cheesy material on the point of breaking down, (203) or at one part of the cavity there may be some evidence of healing and formation of fibrous tissue while destruction of lung goes on at another part. From walls of the cavity a secretion consisting of broken down softened caseated lung takes place, which is expectorated only to be replaced by more as the disease advances. In favourable cases this secretion is gradually arrested and the walls show evidence of fibroid changes, losing their ragged outline and in chronic cases with arrest of disease (319) taking place, the cavity becomes diminished in size, its walls rigid from presence of fibroid material, and may be crossed by fibrous bands, (324) thought by some to be remains of thrombosed blood vessels, by others to be composed of condensed airless lung, (77) the remains of collapsed alveolar tissue originally separating/

separating discrete cavities. As a result of this development of fibroid tissue and its contraction, the size of the cavity diminishes; the chest wall shrinks and alteration in the relative position of the cavity takes place. (7) Addison says that though compound tubercles may of themselves occasionally disintegrate, the chief source is the hepatisation (8) and albuminisation present as a result of inflammatory processes and cavities thus formed frequently show no lining membrane or attempt at repair. The natural cure of the disintegrating process is the formation of a dense lining membrane. (217) Niemeyer thinks cavity formation results from the pressure of cells in chronic catarrhal pneumonia, depriving the alveolar wall of nutriment with resulting caseation, disintegration and cavity formation. (321) A cavity is a source of danger to the sound lung as it is frequently the cause of infection in healthy areas by reinhalation of expectoration, being carried thence through unaffected bronchi, thus setting up a secondary focus of disease. Cavities where the disease undergoes arrest contract and may become completely cicatrised, though partial obliteration is more common. Retraction of cavities/

ies takes place as a result of fibrosis starting from an adherent pleura or developing in interlobular tissue or obsolescent tubercle in cavity wall, and by its shrinking and contraction, the position of the cavity is altered. (322) To fill the vacuum caused by the shrinking and contraction of cavities four changes take place.

1. Emphysema of surrounding lung tissue.

2. Drawing over of opposite lung which may become hypertrophied.

3. Displacement of various organs rarely absent if cavity is large, - the heart, liver, stomach and spleen may have their positions considerably altered as a result of this.

4. Collapse of chest wall.

If displacement of organs and emphysema is insufficient to fill the vacuum, this last takes place. It commences near the clavicles, but overlies a cavity and curvature of spine may result.

The internal changes found in connection with a contracting cavity are:-

1. Retraction of trabeculae which contain elastic tissue from thrombosed vessels.

2. Shortening of bronchi, which become thickened by increase of fibrocellular material/

ial in the peribronchial sheath. This probably acts strongly in drawing the cavity to the root of the lung.

Cavities take months, or even years to contract. The length of Bronchus and freedom for expansion of adjoining lung tissue all affect the probability of the cavity healing. The subclavicular region is the most favourable for this, the sternal region and base less so; secondary cavities rarely, if ever, heal.
(55)

In fibroid phthisis Bronchiectasis is the most active factor in cavity formation. Three factors may enter into its formation:-

1. Shrinking of affected lung by shutting off a certain amount of air space increases the air pressure in unaffected parts and a compensatory dilatation takes place.

2. Contraction of fibroid tissue where it has an attachment to the chest wall and a bronchus draws the bronchial wall out and thus causes dilatation.

3. Secretions may accumulate behind the occlusion in a bronchus.

This form of cavity is lined with a distinct membrane and is usually directly continuous with a bronchus.

In addition to the foregoing changes due to the/

the action of Tubercle Bacilli and other organisms, other changes are found in the lungs and air passages, which are rather the result of altered pressure in the thorax and the shrinking and contraction of areas of lung from fibrosis and cavity formation. In nearly all chronic cases more or less emphysema is found. The contracted condition of the affected lung necessarily leaves an unoccupied space which, the healthy lung having lost part of its natural support and giving way to the increased air pressure fills by becoming emphysematous. (9) Addison says that where tubercles are present in considerable numbers the neighbouring air cells become emphysematous. (323) Williams says that emphysema occurs in connection with chronic tubercular masses in process of arrest with fibrosis, (324) but also in the form of acute vesicular emphysema in miliary tuberculosis. The air passages are also affected by the contraction of the lung. (97) Hamilton quoted by Fagge says that dilatation of bronchioles and bronchiectatic cavities result from stretching of bronchi by fibrous bands from healed tubercles, but Fagge himself doubts this. (10) Addison says that permanent induration of pulmonary tissue/

tissue is occasionally accompanied by dilatation of neighbouring bronchial tubes and that in inflamed tubercular lung, the neighbouring bronchi are dilated with thickened softened parieties. (325) Williams says that in fibroid phthisis there is generally wide dilatation of bronchi, thickening of pleura and interlobular septa. We have seen how this bronchial dilatation may go on to the formation of bronchiectatic cavities in some cases. When the disease is very chronic and becomes partially or entirely arrested, tissue changes of a conservative nature take place. The chief of these is the formation of fibroid tissue which tends also to limit the spread of disease in the lung. These fibroid changes appear also in lungs which are the seat of chronic inflammatory disease, e.g., Pneumonia, Siderosis, even where no tubercle is present, though Moxon quoted by Williams, looked on Fibrosis as the past tense of tubercle.

(11)
Addison held that fibrosis whether associated with Tubercle or not was the result of inflammation of Pulmonary tissue. Niemeyer thought that it occurred in chronic catarrhal pneumonia as a result of the pressure of accumulated cells on the alveolar wall interfering with, but not entirely arresting the circulation/

circulation in alveolar capillaries and as a result of changes consequent on this, the lung tissue became indurated. (326) Williams says it is found chiefly in the alveolar wall of the interlobular tissue. (98) Fagge says the alveolar structure is replaced by fibroid material and that some pathologists say it begins in the Peribronchial tissue spreading to the alveolar tissue. Others again, that it begins in the subpleural and interlobular connective tissue and quotes Willis and Moxon as saying that it begins in the alveolar wall. (327) The fibroid change is always secondary and never primary in lungs, supervening on old pneumonia or tubercle. (328) Williams says a Fibroid lung is reduced in size, dense, tough and cartilanginous in consistence, cavities are cicatrised, caseous masses encapsuled and not uncommonly grey tubercle becomes fibroid. All traces of alveoli disappear and they are replaced by dark grey or black fibrous material into which run white fibrous bands of hypertrophied interlobular tissue. The pleura is thickened and adherent and septa seem to arise from it and from the connective tissue at the root of the lung and bronchi become dilated, forming bronchiectatic cavities.

Another/

Another change found in cases where the disease is chronic and arrested or undergoing arrest, consists in the deposit of lime salts in the affected focus. These cretaceous masses may be found in various parts of the lung, but chiefly occur in the apices in the site of old cavities or caseous tracts, and are generally encapsuled by fibrous tissue. This change is very common in the bronchial glands which have been the seat of tubercular disease. These may ulcerate into a bronchus and be expectorated in the form of chalky sputum.

Situstion of disease in Lung.

Fagge (99) says the upper lobe is most frequently affected. Addison (12) also found indications of inflammation, pneumonic changes and pleuritic adhesions more frequently towards the apex than in the lower portions of the lung. Williams (329) says this is due to defective movement of apex with stagnation of blood in pulmonary vessels and therefore accumulation of exudation. Hamilton and Rindfleisch (100) quoted by Fagge assign as a reason of the upper lobe being most frequently affected, the defective expansion which predisposes to defective circulation/

tion and accumulation of catarrhal products, the defective expansion being due, according to Rind-(101) fleisch, to the upright posture causing the weight of shoulders and arms to fall on the upper ribs and thus interfering with their play. Moxon, on the other hand, says that in the case of patients confined to bed, the anterior edge of the lungs is earliest affected owing to this part being most active during respiration.(24) Clifford Allbut quotes Birsch-Hirschfeld as saying that the reason of this affection of the upper lobe is that the apical bronchus in adults takes a very steep direction upwards, much more so than in children, so that the air stream must be directed to nearly a right angle and that the air current passing outwards has to conflict with stronger currents from the lower lobes so that in expiration there will be a deadpoint in the main apical bronchus near the junction of these streams of air. In weak, flat chests, the apical tubes may sink and further decrease the angle. The most specially affected branches are those between the third and fifth in magnitude in the posterior apical lobe, and here we find the initial changes most frequently take place and from this secondary lymphatic/

lymphatic extension and aspiration infection takes place. From the affected upper lobes extension takes place downwards by continuity and also by aspiration of expectoration. (330) Williams says that cavities are found most frequently at the apex, next, in the dorso-axillary and mammary regions where they are generally caused by inhalation of secretion from apex, the suction being greater in the middle of the lung. Sternal and basal cavities are rare.

PREDISPOSING CAUSES OF PHTHISIS.

An examination of the conditions under which a large number of cases of phthisis develop shows that the phthisis rate varies with the presence or absence of various factors which, however, need not all be in operation at the same time. These factors are shortly:-

Soil Conditions

State of Dwelling-house.
- Dampness
- Defective ventilation and overcrowding
- Situation
- Exposure

Heredity (Consanguinity)

Unhealthy Occupation

Defective Personal Health

Bad Habits and Depressing Surroundings

Contagion.

The special faculty of soil which seems to favour the prevalance of phthisis is its power of retaining moisture. This property is found specially in clay through which water drains away very slowly as compared with a sandy or gravelly soil. The slope of surface also exerts an influence as a permeable soil; sand or gravel, with an impermeable subsoil may become waterlogged if it lies flat and the water is consequently only able to get away slowly, while even a retentive soil gets rid of its moisture much more quickly if it lies on a slope.

A number of enquirers, among other,(147) Bowditch in 1862 in America, Milroy for the large towns of Scotland and Devertie for Sodermanland in basin of Lake Malar or glacial clay, and other observers had noted that the prevalence of phthisis was in direct ratio to the dampness of the ground and Bowditch had noted that draining of the ground is followed by a decrease in the number of cases.(148) Buchanan investigated very thoroughly for Surrey, Kent and Sussex and found that in districts with small phthisis rates, the population resided largely on permeable soil, sand or gravel, while where phthisis prevailed, a large proportion of the population resided/

sided on retentive soil, i.e., a clay soil from which water does not readily escape and also that when an impervious soil slopes, there is less phthisis than when it is level. (331) Williams quotes a marked case of a family in good circumstances with no hereditary disease, but living on a clay soil with a large pond near, where of 12 children, 6 died of tubercular disease, and of 6 living, two suffered from scrofula of spine, and one was delicate. The other three were healthy but had been little at home since growing up. It has also been found that sanitary works with the introduction of drainage pipes over a large area, have in a number of cases reduced the phthisis rate, and where this has failed, (102) Fagge says it is due to the soil having previously contained little water, or by deep drainage being effected by impervious pipes laid in impervious channels so that no extensive soil drainage could take place through or around them. (149) In some towns, Axminster, Ashby de la Zouche, Danzig, after sewers were laid down, the phthisis rate went up 17 - 19% . In these cases probably other etiological factors come into play, e.g., Axminster, where the industry is lacemaking in badly ventilated places.

With/

With regard to Dwelling House, there are three conditions which seem specially to favour the development of phthisis.

1. Dampness
2. Defective ventilation and overcrowding
3. Situation of dwelling as regards exposure to prevailing winds and sun.

We have seen previously that the Tubercle Bacilli grows best in a damp dark place and that according to Ransome the organic exuviae from the body afford sufficient nutriment for it and these conditions by their baneful effect on human health prepare a suitable soil with the leat degree of resistance. These conditions are best found in large industrial centres with their population crowded into one or two-roomed houses in high dwellings which form narrowness of streets, shut out sunlight from each other, and are generally overcrowded and little, if at all, ventilated, in the winter time especially. When the working time is also spent in some indoor occupation as is the case with the large majority, it can easily be seen that the Tubercle Bacilli has in these circumstances most favourable surroundings for/

for its development and growth. Observation also bears out the fact that the phthisis rate is much smaller in agricultural centres than in the large towns and that nomadic tribes living in tents and moving from place to place, enjoy almost complete immunity from phthisis, while those members who exchange the tent for a stone dwelling in towns are apt to develop it.(150) Rash says of last century in United States that phthisis was hardly known amongst those who dwelt on the outskirts of civilization, but was more common in cities and recently Davies has said of United States, that close building and increased population has sent up the phthisis rate alarmingly.

Dampness of Dwelling House, which is an important factor in the production of phthisis, probably acts not only by its being a condition favourable to the growth of Tubercle Bacilli, but also by its being a strong predisposing factor in the production of chest affections. It is seen chiefly in old houses built before the various measures now taken to prevent the entrance of damp had come into use. In the district which I have experience of, partly suburban/

suburban and partly rural, the houses, built within the last thirty years are almost entirely free from damp, while many of the older ones sufrer greatly from it, owing to their floors being often composed of stone flags laid directly on the ground, or if the floor be of wood, there is no provision for ventilation underneath and in many cases, the floor is sunk below the level of the ground at the back of the house, and there is no attempt at drainage to carry water away. In some cases also, the damp seems to come directly through the walls, probably owing to defective lath and plastering. The window accommodation is also generally very deficient, there being often no window, or only a very small one at back which frequently does not open. They consequently miss the aid of the sun and free ventilation which would help to counteract the effects of the damp.

A marked example of the ill effects produced by the above class of house occurred in my own practice. Two small old cottages, lit by small windows in front and one very small window in the back, and standing below the level of the road with a steep

steep/

steep slope down to them and their floors below the level of the ground so that one had to step down on entering, were inhabited by two families, each having several children. In one house, the better of the two, having been to some extent renovated and kept drier by wood boarding round the walls, there were five children and the parents. The other house was very damp, the ceilings leaking and in wet weather pools of water lying at front door above level of inside floors, was inhabited by seven children and parents. In the latter house trouble began with the mother who had an attack of Pleurisy with effusion, from which she recovered; then a few months after, a child developed meningitis which was quickly fatal, and while nursing the child, the mother developed acute phthisis, fatal in a few months. During the winter while the mother lay ill, two of the children had pleurisy with effusion, from which they recovered and remain well to this day. A third had dry pleurisy and also recovered completely. After the mother's death, two of the children left home to work, one in service, another a joiner, in other towns, thus diminishing the overcrowding. Next winter, however, the father/

father, a tramway inspector, who was away most of his time, but slept at home, also developed pleurisy with effusion, but recovered in Hospital and has remained well since - 5 years. Another daughter now left home and they remained healthy for two or three years, when the child who had had dry Pleurisy during the mother's illness and who had been working as a sempstress in a large establishment, developed phthisis and died after a few months' illness. Since then, the remaining children, three in number and father, have remained healthy, but in the interval have been well educated in open air principles. In the first house, at the same time as the mother in the house just alluded to lay ill with phthisis, a child developed broncho-pneumonia and after being ill all winter recovered and kept fairly well during the summer, but in the next autumn she developed tubercular disease of knee joint followed in a few weeks by meningitis and death. No other cases have, however, occurred in that family.

An illustration of the bad effects of the above type of house in the production and spread of phthisis without any intervening chest affection, is afforded by the history of a family in a country hamlet/

hamlet. The house was small, floor on a level with outside ground, small windows in front of house and one very small one in the back, the ground extending up the back wall of the house above the level of the floor. The house was three roomed, but inhabited by a large family - father, mother and eight children, so that there was considerable overcrowding. The mother, a strong, stout woman, developed phthisis and lingered for two years. In the later stages of her illness, a delicate daughter, who had been in service, came home and soon developed phthisis and while she was lying ill after the mother's death, a second daughter developed it and a third had chronic cough and her expectoration showed Tubercle Bacilli. Both the phthisical cases died. The third daughter is still alive, now five years after her sisters' deaths, and has put on flesh though she has still some cough. She has no physical signs and at the last examination, her sputum showed no Tubercle Bacilli. The house was gutted, cement-floored, ceiling raised and windows enlarged, but notwithstanding this, a young brother of 18 developed phthisis about two years afterwards and died after a few months illness. For the last

two/

two years no fresh cases have occurred but the older members of the family have gone to work away from home so that there is more cubic space for each and the ventilation of the house is better attended to. The above case shows the favouring effect of bad housing, especially coupled with overcrowding on the incidence and also the spread of phthisis.

Defective Ventilation and Overcrowding.

All authorities are agreed that living in an atmosphere rendered foul by overcrowding or defective ventilation is a direct cause of phthisis. (151) Hirsch quotes Clark and Flint to show that occupation effects the phthisis rate in so far as it compels to a sedentary life with insufficient fresh air and excercise, and (152) Simon, as concluding from Greenhow's enquiry that a high phthisis death rate occurred in the male or female population according as either followed an indoor occupation. Smith at Brompton found that out of 1000 persons treated for phthisis, 70% have been in the habit of spending their time in overcrowded, hot and dusty places indoors. (153) Poulet notes that in Planchet - les - mines the/

the change from an agricultural to an industrial centre caused a rise in the phthisis rate from a practically unknown quantity to 12.5% of the total mortality. It is found also in the army that phthisis is much more common in barracks than when soldiers are leading an active open air life.(154) Welsh says that nearly half army consumption is connected with a vitiated barrack atmosphere.(332) Williams ascertained that of 3214 at Brompton, 1812 followed indoor occupations, working largely in ill-ventilated rooms.(103) Fagge quotes the Royal Commission on the sanitary condition of the army in 1858 as showing that the excessive death rate in the army from phthisis, rising in the Foot Guards to double that of civilians, was due to deficient and defective accommodation and the introduction of sanitary improvements was followed by a fall in the phthisis rate.(104) He quotes from Parkes' Hygiene, "that in Leopoldstadt Prison (Berlin?) which is badly ventilated, out of 86 per 1000 deaths 51.4 per 1000 were from phthisis, while in the well ventilated House of Correction in the same city, only 7.9 per 1000 died of phthisis, the diet and mode of life being the same in each establishment."(155) Hirsch corroborates these facts from Millbank Penitentiary and the Prisons in Lower Bengal.

In/

In ordinary country practice one generally finds overcrowding and defective ventilation associated with other causes - damp dwellings and defective nourishment, as it generally means a large family in a small house. In one family, however, which I attended, the two latter conditions could be excluded, as the house was on a second story and indeed could hardly be said to be overcrowded, but was decidedly badly ventilated and as the occupants worked in the house as sempstresses and were little out of the atmosphere, I think this occupation and the constant living in a badly ventilated atmosphere were, setting aside the influence of heredity, the chief causes of the disastrous results to the family. The family consisted of a father, a joiner, ill with chronic phthisis, the mother, a strong woman who was never affected, in any way, a son, who married and went out of the house shortly after his father's death, a daughter in service and consequently never in the house, except perhaps for an hour or two once a week or so and three daughters who lived at home for a little before the father's death. The son and daughter who went out of the house are still alive and healthy. Of the other three, the/

the eldest a nurse, had influenza while attending a case and developed phthisis after that, coming home with the disease on her. She improved a little and after her father's death , lived at home except for a few weeks when she was in the Infirmary. The other two were healthy at the father's death, but during the next winter (i.e., within a few months during which they were constantly at home), they both developed phthisis, the younger one dying in a few months but sewing up to shortly before her death. The other one offered more resistance and keeping fairly well, with her other sister continued sewing steadily at home, but in twelve months afterwards they both died, one of gradual exhaustion, the other suddenly of haemo-physis. I cannot help thinking that if the two younger sisters had been living out of door lives or had been able to live out of the house altogether they might not have developed the disease, though of course, the occupation or house cannot be blamed for the elder sisters developing it, as she came home from her nursing with definite evidence of the disease on her.

Heredity/

Heredity.

It was at one time thought that the disease itself was inherited, but there are only a few cases on record of a child being born with tubercular disease developed. (63) Cohnheim says that the few observed cases of congenital tuberculosis, favour the view that it is an infective disease which, like syphilis may be communicated from parent to child. He thinks, however, that the subsequent development in children of afrected parents may be as well explained by contagion and favouring conditions, as by infection from parent, or inherited predisposition. (333) Williams quotes the case observed by Sir C. Scudamore of an infant born with developed tubercular disease and says (334) that in the case of the marriage of consumptive and healthy person the offspring may become tubercular and if after death of consumptive parent, the healthy one marries again, the offspring of this union are healthy. (218) Niemeyer thinks that what is transmitted is a weakness or vulnerability of constitution. (156) Hirsch says that the occurrence of tubercle in families from generation to generation is frequently seen, but what is transmitted is a defect in the organisation of the respiratory system/

system predisposing to the disease. Heredity occurs in 33-40% of all cases of phthisis.(105) Fagge thinks a vulnerability is transmitted, but this can be kept in abeyance by hygienic precautions.(335) Williams quotes statistics from the Brompton Hospital Reports collected by Fuller (59%), Cotton(36.7%) Pollok (30%) and himself, and finds the percentage of heredity to vary considerably according to the relatives included, from 12% where parents only are reckoned to 48.4% where parents, grandparents,(336) brothers, sisters, uncles, aunts and cousins are included and finds that heredity affects women more than men in the proportion of 57-43%.(337) R. Thompson from a careful analysis of 80 families with hereditary disease concluded that the male had more power of resistance than the female.(338) Transmission was more common through the mother than the father, mother's influence being exerted both at conception and during gestation. Fathers transmit more frequently to sons and mothers to daughters than vice versa.(339) Maternal inheritance is worse for both cases, while paternal seems to increase the resisting power of daughters.(340) Double hereditary hastens the onset of the attack and renders it more fatal.

Williams/

(341) Williams thinks that family predisposition hurries the onset of the disease and that the children die at an earlier stage than the parents. (342)

(25) Davies, in an article on phthisis in the Isle of Man where intermarriage is common, the people generally marrying in their own district, finds that consanguinity of marriage is a strong predisposing cause.

Out of 32 cases I have collected, I find family predisposition in 19 cases. Eleven cases out of 9 families showed a history in some ancestors, either parents, uncles, aunts or cousins and 8 other cases out of 7 families showed a history of disease in brothers or sisters. I have found it difficult to trace heredity among working-class patients and inaccuracies are apt to creep in owing to the confusion of other chest affections with phthisis, but I think the above figures show that there is at least some weakness transmitted which favours the onset of the disease. The following family history is, I think, strong evidence of the transmission of vulnerability. The parents were healthy, but one cousin and some relatives of the parents died of phthisis. The disease showed itself first in the case of a daughter who died of it in 1889. About

a/

a year after, a son developed phthisis and died after two years' illness; some months after a second daughter developed the disease and about fifteen months after she died, a third. These all developed the disease consecutively, the one being dead before the next showed symptoms. A second brother who was married and lived out of the house, having little or no communication with the family and having fallen on bad times, also developed the disease. There was too long an interval between the cases at home to make contagion a strong element in the case and their home surroundings were good, so that there must have been a transmitted vulnerability to account for so many members of one family developing the disease. I have seen four mothers suffering from phthisis themselves, bear children and of these children all healthy at birth. One died of meningitis when eight months old, the disease being then quiescent in the mother though she died of it several years afterwards - the infant was bottle fed. A second, whose mother was just showing the initial symptoms and died two years afterwards, had measles when about twelve months old and had been strumous ever since, suffering after/

after the measles from suppurating bronchial gland, eczema, diarrhoea and phlyctenular ophthalmia. When one condition yielded to remedies, another developed. The child is now at four years old stronger, but still has attacks of phlyctenular ophthalmia.. The third in whose mother the disease was chronic, was a very ill-thriven child at birth, but developed no actual disease up to 18 months of age when they left the district. The fourth whose mother had been phthisical for about two years before it was born, is now 5 or 6 years old and has been and is quite healthy. In a fifth case, the mother developed acute phthisis a few months after the child was born and quickly went down. The child died of meningitis at 18 months of age. All the children were bottle fed. In another case where the mother was healthy, the father had slight evidence of disease, but with little activity of it, the first child died at three years of age of Bronchitis and albuminuria, while a second born when the first was two years old was healthy. They left the district after the father's death, when the child was about 18 months old. The above cases are strong evidence that/

that though the disease itself is not transmitted, still there is a great susceptibility to it handed down.

Another interesting case would seem to suggest that a parent, who only developed the disease when the child was some years of age and probably largely through bad surroundings, might transmit a vulnerability to his child. The family consisted of a father, mother and one girl. When the girl was 12 years old and had gone out of the house into service, so that she was largely removed from the bad home surroundings and contagion, the father who had been at one time a hard drinker, had influenza and getting out of work was badly fed. Phthisis then gradually developed and ran a chronic course, proving fatal in about four years. During this time, the girl was out at service and he was nursed by his wife who slept with him. After he died the girl came home with apparently nothing more than a bad cold, and recovered so far as to return to her place, but she never felt fit for work and in a few weeks returned home with the physical signs of early phthisis, while the wife who had had very ample opportunities of being infected continues well 11 months after her husband's death.

Dusty Trades and Occupations.

There are certain trades which owing to the irritating particles being given off in the working directly irritate the respiratory passages and lung, and predispose to phthisis. These trades are stone masons, millstone workers, needle grinders, potters, etc. Authorities agree as to the effect produced on the lungs by inhalation of these irritating particles, but while some think that the changes so produced are different to and independent of tubercular disease, others incline to the belief that the Tubercle Bacilli played part in these cases also.(219) Niemeyer cites these trades as an important cause of phthisis.(343) Williams says the presence of Tubercle Bacilli and Tubercles along with cavities shows the tubercular element in these cases also.(106) Fagge declines to accept the opinion that pneumo-conioses are essentially different from phthisis.(107) He holds that they bear a strong resemblance to fibroid phthisis and says that though undoubted tubercles have not been described, still there are almost invariably found nodules which in size and appearance resemble tubercles which have undergone fibroid transformation(108)/

tion and also emphasises the fact that the course of the disease in the lungs resembles that of tubercular affections, i,e., one lung is generally attacked before the other and the upper lobe is almost invariably attacked first with extension downwards to base.

(273)
Rindfleisch says of Siderosis, that in addition to catarrhal changes in Bronchi and overgrowth of interstitial connective tissue, there are scattered through the entire organ, tough greyish transparent nodules varying in size from a pinshead to a pea which clearly resemble (274) localised tuberculosis.

(56)
Coates, on the other hand says that though true tuberculosis may possibly be induced by inhalation of irritating dust, the great majority of lung diseases from this cause differ from tubercular phthisis. There is no tubercle formation, seldom cavity formation except by bronchiectasis and little constitutional disturbance. Patient can generally work on for years till dyspnoea incapacitates him.

Contagious Transmission of Phthisis.

This subject has during the last few years been very prominently in the foreground as the main cause of the propagation of phthisis.(109) Phthisis had, however, been regarded in Italy for long as a contagious disease and in this country various observers had come to the conclusion that contagion might play a part in the spread of phthisis under special circumstances.(157) Humphrey of Cambridge and Mahomed of London, who edited the Collective Investigation Report on the subject, concluded that if phthisis be communicable, it is only under circumstances and conditions of extremely close personal intimacy, e.g., persons sharing the same bed or room or shut up together in numbers in badly ventilated rooms.(158) C. J. Williams says of Brompton that among the physiciams or assistants, clinical clerks, nurses, etc., (many of whom lived continuously in hospital for years) phthisis has not claimed more than an average share of victims as compared with the civil population of towns, and only in three or four cases could it be brought into connection with individuals resident in hospital and that in spite of sputum only being disinfected when disagreeable/

disagreeable odour present, although the jars were emptied two or three times daily and there was defective ventilation as evidenced by erysipelas and sore throat.(159) He admits that in private, a few cases of phthisis had occurred among those in close association with consumptives, but bearing in mind the number of cases where no spread of phthisis occurred, the negative evidence would seem to preponderate over the few positive examples.

(110) Vertue Edwards in 1867 after being 17 years resident medical officer at Brompton, stated that he remembered 59 residents with an average of six months and of these only three were consumptive and of many nurses with a residence varying from months to 12 and 24 years, the head ones sleeping in a ward of 50 patients, only one was known to have died of phthisis and that after an unhappy marriage.

(111) Dr Weber in the Clinical Society's Transactions for 1874 recorded some cases where the disease seemed to spread from husband to wife. Nine husbands affected with phthisis had 18 wives. One lost 4 wives in succession; one lost 3; one lost 2, and three had one wife each. Only one of the wives who were/

were all healthy at marriage had a tubercular taint. All the husbands outlived their wives, who all bore children and Weber thinks that the absorption of semen was the cause of the transference of the disease. (112) Fagge, however, thinks that the transference of the disease was not actual, but due to the inexplicable influence of impregnation. Weber only knew 30 other phthisical husbands whose wives escaped, while on the other hand of 29 consumptive wives who married, only one lost a husband.

Fraser of Tynedale reports 26 fatal cases where either husband or wife was affected and the couple shared the same bed and lived in close intercourse and no transmission of disease took place. (160) Reginald Thomson observed 15 well marked cases of wives infected by husbands out of 15,000 cases of phthisis, Williams concludes that the evidence of large consumptive hospitals shows that phthisis is not a distinctly infective disease like a zymotic fever and is not in the ordinary sense of the term an infective disease, as, though the opportunity of contagion frequently occurs, instances are rare; when it does occur, the surroundings are found to be specially favourable/ (344)

favourable for it and he thinks that by attention to ventilation, separation of consumptives at night and the milk supply, it can be avoided. (64) Cohnheim "Thinks that the fact of several members of a family becoming tuberculous only shows the presence of favourable conditions." In every-day practice one sees cases which seem to justify both sides of the question. As far as my personal experience goes, I find that where more than one member of a family developes phthisis within a short time of each other, they are almost invariably living under unhealthy conditions. I have only seen one case in which more than one member of a family was concurrently affected when the sanitary and hygienic surroundings were good and in this case, the possible infecting case showed no active symptoms of the disease, but had previously had two or three attacks of haemophysis and developed acute phthisis twelve months after, while the patient himself, a child of seven years old , had been a frequent sufferer from bronchitis and developed the phthisis after an attack of influenza. I have, however, seen in one family of middle-class position one patient after another sicken/

sicken and die of the disease, but no two were ill concurrently. Whether these are cases of infection from one to the other where the germs lie latent, or at all events, cause little damage at the time, or merely cases of outside infection occurring in those with an inherited weakness of respiratory system, it is doubtful. But on the other hand, in the case of families in bad hygienic surroundings, I have seen several undoubted cases of infection. In one case, a damp, badly lit and ventilated house was occupied by a large family which must have overcrowded it considerably. The mother developed phthisis and after a time while she was still alive, first one daughter and then another developed the disease, while a third lost flesh and developed cough and had Tubercle Bacilli in her sputum without, however, having definite physical signs in her chest. She is still alive and has put on flesh again and enjoys better health, but in the interval between the death of the sisters and the present time, a brother developed the disease and died in a few months. The house, was however, renovated and much improved before he took ill, and the occupants fewer in number owing to the above deaths/

deaths and some of the elder members of the family leaving home to work. No further cases have occurred for three years now. In another case, the house was dry and had large windows, but three sisters were constantly in it sewing and for this purpose occupied a small room with the window closed. One was a victim of the disease and the other two developed it, all three dying within a few months of each other. These two instances seem to me to afford evidence of the probability of infection taking place in unhealthy surroundings, where the members of the family are much in contact.

The following cases, on the other hand, show that frequently no infection takes place even on the closest contact and where the surroundings are by no means first class.

In four cases the wife was affected and the husband, partly to attend to his wife and also for lack of other accommodation, as they all occurred in working-class families, occupied the same bed as the wife during the whole course of the illness and yet contracted no disease. In other two cases, the husband was affected and the wife slept in the same/

same bed and yet escaped infection. One of these cases was specially interesting from the fact that the wife bore a child conceived during her husband's illness and yet she remained alive and healthy some years after her husband's death. These observations agree entirely with those of Willaims and others, that it is only where the circumstances are unfavourable to general health and where the person is in constant association with the phthisical patient that infection takes place and even where circumstances are favourable, it frequently does not occur. In the above cases of husbands and wives, five out of six of the unaffected members were away at work the whole day in good conditions as to fresh air.

Defective Personal Health.

(161) Hirsch says that all things detrimental to the nutrition of the organism in general, making it vulnerable through defective repair in tissues, predispose to morbid processes in general and phthisis in particular. This explains its development in illnourished people and those worn out by severe sickness, diabetes, tabes dorsalis etc. (220) Niemeyer says prolonged suckling, diabetes, insufficient food, mental depression, etc., are causes of Tubercle

Fagge/

(113)
Fagge says that the importance of a badly formed chest in relation to phthisis may be merely due to its being an indication of defective bodily development. Diminished resistance is found in those whose bodily health is enfeebled by worry and excessive work, insufficient exercise, fresh air and
(204)
food. Koch emphasises the greater danger of mouth breathers to infection by inhalation of Tubercle Bacilli. In nose breathers, the bacilli are largely arrested by the nasal mucous membrane. Mouth breathers have no such safeguard and Tubercle Bacilli get more readily into Trachea and Bronchial tubes, but if the ciliated epithelium is healthy, they will probably be directed upwards to the surface again.
(205)
On the other hand, if there happens to be a spot denuded of, or with weakened epithelium, e.g., such as may be found after measles, whooping cough, etc., the Tubercle Bacilli get a good chance of establishing themselves.
(114)
There is a form of chest due to imperfect development, which not frequently gives rise to caseous degeneration and tuberculosis of lungs. It is characterised by long narrow and shallow chest, sloping Supra and Infraclavic regions wide intercostal spaces, wing-like projection of scapulae/

scapulae, prominence of acromial ends of clavicles, increase of angle between manubrium and body of sternum and diminution of antero-posterior diameter. This form of chest predisposes to phthisis, though by living carefully many people of this conformation escape the disease. In many cases of phthisis one gets a previous history of chest affection. In 35 cases, I have examined personally, twelve had at one time of their lives suffered from some chest affection. Of these, five had had pleurisy with effusion at intervals of twelve months in two cases, 1½ years, 3 years and 12 years in the other elapsing before phthisis developed; one had an attack of dry pleurisy four years before phthisis developed. One suffered from asthma and bronchitis in childhood and youth. One frequently had bronchitis in childhood and developed phthisis at 7 years of age. One had, in addition to frequent bad attacks of bronchitis in childhood, a bad attack of broncho-pneumonia a few months before phthisical symptoms developed. Two had pneumonia (croupous) several years previous to phthisis and one had croupous pneumonia twelve months before.

Williams/

(345)
Williams says out of 1,000 cases of phthisis in 267 bronchitis and pleuro-pneumonia preceded the disease. Influenza has been the apparent exciting cause in a few cases, the patient never completely recovering, but drifting into phthisis without any definite recovery in the interval. In one or two cases, nursing seemed to be the only predisposing cause at work. I have seen one case in which it followed measles in a young lady and in one case in which the disease was running a subacute course, an attack of measles was followed by an acute outbreak which speedily terminated in death. In one chronic case, a mild attack of measles did not influence the course at all. I have seen it follow whooping cough in the adult in the case of a man in the prime of life, perhaps however, weakened by previous malarial attacks.

Bad Habits and Depressing Surroundings.

These may indeed be considered along with the above, as they act in the same way by diminishing the resisting powers of the system. (346) Williams says mental depression is frequently followed by irregular habits. Food is taken irregularly and insufficiently/

ficiently and stimulants are apt to be resorted to. He quotes Laennec's instance of a religious association of women whose rules were very austere and who were being constantly tried by opposition and contrariety to make them renounce their will. In a few months they developed phthisis and in ten years the institution was cleared out two or three times, a small number only escaping, and these were distracted from their religious tasks having to go into the city frequently on business. The diet was ascetic and severe.

Alcohol.

(347) This bulks largely as a predisposing cause. Inability to take sufficient food is soon followed by wasting and loss of power to resist weather and a slight catarrh leads to tuberculisation and rapid excavation of lung. I have seen only three cases out of between 30 and 40 observed, in which alcohol could be traced as a definite exiciting cause. In the first, the patient had been a heavy drinker for years and in his case both lungs were invaded and he succumbed after a few weeks' illness. In the second the patient had gone in for heavy bouts of drinking alternated/

alternated with periods of sobriety and previous to developing phthisis he had suffered from influenza and owing to his alcoholic habits had been living in great poverty, an indirect effect of the alcohol which would also predispose to the disease. The third case occurred in a young man of about 35 years of age, a well-to-do grocer, who lived in good surroundings, but owing to his taking daily more than was good for him, had brought his stomach into such a state that he could take little nourishment. He suffered one spring from pleurisy with effusion from which he made a good recovery and put on flesh again and got much stronger, but never entirely lost cough. He would not, however, follow advice as to his drinking habits and in the ensuing spring got his stomach into such a condition again that he vomited everything; the cough got worse, he lost flesh and now evidence of tubercular disease was evident in his lungs. He made a partial recovery during the summer when his amount of drink was strictly controlled, as it was found that unless he got a small quantity he would not eat. However, I am afraid the ultimate result of the case will be bad, as now that/

that he is stronger, he goes to his shop and almost invariably gets an extra supply of liquor.

(26)
Brouardel quotes Beaudran of Beauvais results of comparative effect of alcohol on the phthisis rate:-

Deaths from Tuberculosis in 10,000 inhabitants.	Annual Consumption of Litres of Alcohol per head.
30 - 40	12.47
40 - 50	15.41
50 - 60	14.72
70 - 80	16.36
80 - 90	17.16
more than 90	50.70

SYMPTOMS OF PHTHISIS.

The onset of pulmonary phthisis presents different aspects according to the degree of resistance which the organism is able to make to the Tubercle Bacilli, to the presence or absence of other micro-organisms and the dose of the poison admitted into the system. It is insidious in onset and slow in development/

development in some, in others it begins suddenly and hastens to a speedy termination, while in others again, it pursues an intermediate subacute course . In Chronic Phthisis i.e., cases in which life is prolonged for a number of years, the disease generally first manifests itself by cough with a little purulent or muco-purulent expectoration. In some cases, the cough is violent and spasmodic and produces vomiting even in early stages and in one case the patient vomited his food for some months before the cough developed or the disease was evidently phthisis. The cough in some cases for the first year or two disappears during the summer only to return next winter and soon it persists all the year round. After the above symptoms have persisted for a longer or shorter time, they are followed by loss of appetite, strength and flesh. There may as yet be no evening rise of temperature, little or nothing in the way of physical signs, and the diagnosis in the absence of Tubercle Bacilli in sputum rests mainly on the association and persistence of the above symptoms. As the disease advances, the expectoration increases in amount, the loss of flesh/

flesh becomes more marked, breathlessness on exertion appears and the patient becomes anaemic, and if a woman amenorrhoeic, the finger-nails begin to get clubbed and there may be come cyanosis and may be tendency to night sweats and a slight rise of evening temperature. In some cases haemorrhage may come on, and indeed, may be one of the earliest symptoms and occasional pleuritic pains also manifest themselves. As the disease progresses the symptoms become more marked. Haemorrhage, if it does occur, is more severe; emaciation becomes very marked, the temperature assumes a hectic character, night sweats become severe and occur also if patient sleeps during the day. Ankles may be a little puffy and more or less intractable diarrhoea appears. Albuminuria is present in some cases, and sometimes the mouth and fauces show a number of yellow ophthous patches of a painful nature and in the last few days there is often a considerable development of thrush in the mouth.

Subacute Phthisis.

In a considerable number of cases the first onset of the disease is accompanied by a moderate amount/

amount of fever with cough and expectoration of a frothy and muco-purulent character. The lung shows patches of broncho-pneumonia and tubercle bacilli are found in the sputum. Possibly with rest and treatment the symptoms may subside for a time, but in many cases they recur again, sometimes with an attack of haemorrhage. The lung has never cleared up in spite of quiescence of symptoms and now shows renewed activity and fresh areas of infection. The temperature again rises. Pulse becomes quicker, loss of flesh occurs, night-sweats appear, cough and expectoration are worse; some cyanosis and diarrhoea appear and the patient runs down in a year or less after the first symptoms have appeared.

Acute Phthisis.

Here the patient is obviously very ill from the first. There may be a feeling of shiveriness and associated with this, a high temperature. In one case under my observation the only symptoms at first were the above. The temperature was 104° in the middle of the day, no cough or expectoration and on two occasions before the lung condition was evident, the temperature went temporarily to 107° and 109° /

109° in the evening. Shortly, cough appeared and then a little frothy muco-purulent spit; physical signs become evident in the lung, night sweats, loss of flesh and diarrhoea rapidly followed and the patient died after two months' illness.

In another case, the disease began with haemophysis followed by moderate fever. Both had subsided and the patient appeared to be doing well, when one day after straining at stool he had a violent rigor followed by high fever and frequent pulse and rapid consolidation of one base. In spite of all that could be done the disease ran a rapid course. The cough was never very troublesome and not much expectoration, what there was being chiefly frothy and slightly muco-purulent in character. Sweating, diarrhoea and rapid loss of flesh occurred and the patient died about six weeks after the onset of the disease.

Miliary Tuberculosis of Lungs.

In this form of the disease it is sometimes difficult to absolutely distinguish from typhoid fever. There is considerable fever, rapid wasting, great dyspnoea, frequent pulse, cyanosis and little cough/

cough or expectoration. Death occurs after a short illness. Some or the symptoms require a little further consideration.

Haemoptysis.

This may occur at any stage of the disease and so frequently before there are any other evidences of the disease that the haemorrhage was thought by Niemeyer (221) to proceed from the bronchial mucous membrane (222) and being inhaled into the lung to be an exciting cause of phthisis. In the early stages, Coates (57) says the blood escapes by diapedesis from the pulmonary capillaries, while in the severe haemorrhages of the later stages, it results from rupture of a branch of the pulmonary artery in the wall of a cavity (348). It is due to fatty degeneration, ulceration and erosin of pulmonary vessels during tubercular changes (349) and in the severe cases from aneurism (350) of a branch of pulmonary artery. In some cases it is followed by no febrile state whatever and the patient seems none the worse, while in other cases there is considerable fever, associated with temporary extension of the physical signs in the lung, inflammatory, or in some cases due to fresh foci of disease/

disease resulting from Tubercle Bacilli inhaled with the blood. Sometimes the extension of disease along with the symptoms induced by it continue until the patient, who may have been fairly well before the onset of haemorrhage, succumbs to the disease in a comparatively short time. In other cases, after an illness of a few weeks, the patient regains his lost ground. Sometimes the vessel affected is so large and haemorrhage so severe that sudden death takes place, even in patient in good condition and apparently with a good spell of life in front of them.

Pyrexia.

This is due to at least two different causes.

1. Spread of disease in lung - tuberculisation and inflammation.

2. Absorption of poisonous material from the decomposing discharges of cavities - septic poisoning.

In chronic cases, with the exception of occasional exacerbations, the temperature may show little divergence from the normal. It is as a rule rather below normal in the morning and slightly above in the afternoon and evening, until as the disease/

disease progresses, hectic fever begins to manifest itself, though in some cases a subnormal temperature persists in the later stages of the disease also, perhaps going up to 99°F. or thereabouts at times. In one patient, I observed the temperature closely for several months, the case was of two years' duration, with bad cough, but no expectoration, and disease evidently slowly advancing in lung which also contained a cavity. He was keeping up his flesh fairly well, taking nourishment fairly and mostly in bed. His temperature ran as follows:-

9 a.m. ..... Sub-normal - 99°F.

2 p.m. ..... 99° - 100° or 101° F.

6 p.m. ..... 100° - 101° F.

9-10 p.m. ... 100° - 101° F.

The last temperature was sometimes a degree higher than the 6 p.m., sometimes a degree lower. There were temporary exacerbations when the highest temperature would be 102° at 6 p.m., with a drop to 101° at 10 p.m., or again, the highest record during the 24 hours might be 100° , but the above figures were a fair average. This closely resembles the temperature/

ature given by Williams for first stage and second stage phthisis with cavity forming. In these, Williams indicates a slightly subnormal temperature at 8 a.m., rising to a slightly feverish condition (99.3 mean temperature averaged from number of cases) about 2 p.m., and a gentle rise continuing till 8 p.m. (mean temperature 99.6) after which temperature fell to normal at 11 p.m., and subnormal in early morning in the first stage and in the second stage, the temperature ranges somewhat higher and the rise is more continuous from 8 a.m., to 8 p.m., beginning at 11 a.m.

In my case, the highest record was sometimes at 10 p.m.; at others the fall had begun to take place then. With a freely discharging cavity, the swing of temperature is much more marked. According to Williams the temperature may rise from a minimum of 91.6 in the morning to a maximum of 104.6 in the evening. He says the temperature begins to rise at 10 a.m. and continues doing so steadily till 10 p.m. When the average maximum is obtained, then the fall begins; at first slight, but getting considerably below normal in the early hours/

hours of the morning.(359) This temperature resembles that in chronic abscess and is therefore probably due to suppurative process.(360) Wunderlich says in acute phthisis the daily difference may amount to 5.4° F. The daily maximum is generally in the afternoon or evening, but may be found in the morning, as I have seen in one case of pulmonary phthisis and psoas abscess, where the highest temperature was recorded in the morning with a corresponding fall in the evening. In acute and subacute cases, the temperature may be above normal both morning and evening, but there may be an exacerbation in the afternoon and evening. The course of the temperature may, however, be temporarily modified by various circumstances. Physical exertion is, in some cases, especially in those only taking occasional exercise, followed by a temporary fall complicated, however, by a higher rise than usual after resting later on in the afternoon. Exhausting conditions of any kind, e.g., severe diarrhoea whether temporary and of mycotic origin, or coming on towards the close of the disease with waxy changes and tubercular ulceration cause a low, frequently subnormal temperature for the time being. The occurrence of meningitis/

meningitis in old standing cases also causes this, as in one case where there had been steady rise of temperature and frequent pulse for months, after a few days of headache and sickness the temperature became subnormal and the pulse slow. In patients of good vitality, it may be temporarily elevated by excitement or over much exercise short of fatigue however.

Diarrhoea is almost always found in the later stages of phthisis.(360) In the Brompton Hospital Reports for two different years, tubercular ulceration of the intestines occurred in 70 - 80%.(361) The ulceration begins with inflammatory hyperplasia of solitary and agminate glands in the lower part of the ileum and extends to large intestine. Caseation and suppuration occurs and an ulcer with smooth floor and sharp cut edges results.(275) Rindfleisch says the tubercular element is quite subordinate in tuberculosis of intestines, the primary change which occurs in Peyer's patches and solitary follicles(276) consisting in proliferation of the corpuscular elements,(277) with subsequent caseation and ulceration, but presenting no evidence of any specific agent.

Miliary/

Miliary tubercles, however, develop on branches of vessels and lymphatics and determine the direction of extension of ulcer which is therefore chiefly transverse and extends beyond the limits of Peyer's patch. (362) The muscular coat of the intestinal wall gets eaten through and the peritoneal coat thickened. Perforation may occur and peritonitis be set up. (278) Rindfleisch notes a case where five tubercular ulcers of the ilium had perforated into other parts of the bowel. Severe haemorrhage may also occur. Secondary to this ulceration, the mesenteric glands may become enlarged. The diarrhoea is severe and persistent. It is due to swallowing expectoration and frequently occurs in laryngeal cases and in weakly people unable to expectorate properly and may also result from ingestion of tuberculous milk or meat. In protracted cases the diarrhoea may also result from lardaceous disease of intestine.

Albuminuria.

I have only rarely found this in uncomplicated pulmonary phthisis, possibly owing to so many of my cases running a subacute course and terminating after/

after about a year's illness. I had an example of it in a girl of 17 years, who had been ill for 15 months, during which she had a period of temporary quiescence lasting for several months, when she put on flesh and lost her cough and spit. The disease, however, again became active and in a few weeks intractable diarrhoea supervened. Her feet and legs became dropsical and an examination of her urine showed the presence of a large quantity of albumen with the presence of granular and epithelial casts. Death occurred in a few weeks. In another case, an adult with psoas abscess complicating phthisis of several years' duration it was present to a slighter extent. (363) Williams says it occasionally occurs where there has been no exhausting discharge, e.g., diarrhoea or profuse expectoration and that he has seen it disappear, only, however, to return with resulting death of patient. He says that the changes in the kidneys are in the majority of cases of a lardaceous character (364) (in 133 necropsics recorded in Brompton Hospital Reports for one year and a half 52% showed evidence of lardaceous disease), and quotes Bamberger as saying that parenchymatous /

parenchymatous and tubular forms of kidney disease were most common. There was diminution of water and urea, but as there was diarrhoea present, he thinks that this would partly, but not entirely, account for the deficiency.

COMPLICATIONS OF PHTHISIS.

Laryngeal Phthisis.

This is fairly common. In 32 cases of phthisis under my observation laryngeal phthisis occurred in six. It is due to infection of larynx from the lung by the sputum in the majority of cases at least. I have never seen it without previous disease in the lung. It first shows itself by huskiness of the voice and later on may give rise to a great deal of pain, making swallowing extremely difficult.

Laryngoscopically.

At first the appearance which engages attention is general congestion, followed by thickening in arytenoid regions and interarytenoid growths, then the false cords become thickened and ulceration of true cords and epiglottis also occur.

Vertebral Abscess.

I have seen once occurring in a case of chronic phthisis of several years' standing - it was preceded for two months by neuralgic pains of sciatic nerve and hastened end by its weakening discharge - the patient only living about two months after it was opened.

Fistula in Ano.

Occurred three times in 32 cases, but was left alone as the patient was by that time manifestly unfit for operation.

Meningitis.

Occurred in one case out of 32; its onset heralded by severe headache and vomiting; subnormal temperature, slow pulse, coma, partial paralysis and death in ten days after its onset. The patient had been about eight months ill before it developed.

Pneumo-thorax.

Also occurred in only one case out of 32. The patient had been nine months ill and the disease was far advanced in the lungs and he was going down quickly. Its onset was betokened by severe pain/

pain in the side, increased dyspnoea, bad cough but diminution and arrest of expectoration, and the physical signs of the condition. Death resulted in five days after its appearance and was probably hastened by it.

PHYSICAL SIGNS OF PHTHISIS.

The earliest evidence of tubercular disease in the lungs is generally found in the upper parts. (279) Here palpation may show slight lagging of one or other infraclavicular region behind the other in respiration. (115) The percussion sound may be healthy owing to the diseased areas being very small or being surrounded with healthy lung, (280) or it may be possible by mapping out both apices to show that the diseased one is contracted as compared with the other, or owing to the consolidated areas being mingled with tissue, slightly relaxed, but air containing, there may be a tympanitic deadened sound. (116) (117) Auscultation shows rough or jerky respiration at apex with prolonged expiration and perhaps a few râles. (281) In some cases pleuritic friction is the first indication of the lung affection. As the disease/

disease gains ground and the consolidation increases in amount, the signs become more marked.(282) The defective movement, lagging of upper part of one lung may now be evident to inspection alone, and there may be deepening of supraclavic depression,(283) Palpation of intercostal spaces over the upper part of lung may elicit pain due to callous thickening of pleura.(118) The precussion sound is muffled or absolutely dull depending on degree of infiltration and(119) blocking of air cells and the extent of lung involved.(286) This is chiefly found in the supraclavicular, clavicular and supraspinous regions. In cases where it is very marked and accompanied by increased resistance, there is generally pleural thickening.

(285) Frequently in early phthisis there is a transition breath sound, the infiltrated parts of the lung favouring the transmission of bronchial sound unchanged while the parts containing air convey a breath sound to the ear toned down to a vesicular murmur.(286) Over areas of caseous pneumonia râles of a fine crepitant character are heard, chiefly during inspiration or the very beginning of expiration.(120) Where there is a considerable extent of consolida-

tion/

tion bronchial respiration is now heard (121) accompanied by medium sized and coarse râles due to bronchial catarrh, and frequently (122) there are resonant, ringing and consonant râles. (287) Where the consolidation is of the nature of chronic induration, a peculiar creaking or crackling râle is frequently heard. (124) The vocal resonance is broncho-phonic in character. When extensive consolidation is associated with cavitation, (125) inspection of the chest may show circumscribed depressions of surface in the supra and infra-clavicular regions due to shrinking of the lung as a result of caseous condensation. (126) In addition to limited respiratory movement, parts of the chest between 1st and 3rd ribs in front may be seen to sink in inspiration and bulge outwards in expiration. This is due to condensation and cavitation of the subjacent lung, which thus becomes unable to expand to follow the respiratory movements. (127) Where the upper lobes are extensively affected, there is more vigorous action of the lower lobes which become widely distended, while the movements of the upper lobes are restricted. (288) In fibroid phthisis with long continued contraction of one lung/

lung there is shrinking of one side. The spinal column is curved and there is diminution or absence of respiration on the indrawn side, while the healthy side develops a vicarious emphysema. Partial shrinking is, however, more frequent.

(128) The vocal fremitus over the consolidated lung is increased if there is free communication with the trachea. If the bronchus is blocked, the fremitus is decreased. This also occurs in superficial cavities surrounded by dense walls. (129) The percussion note over consolidated lung is dull. (130) If there is a cavity of fair size - as large as a pigeon's egg - with firm walls and situated close to the surface of the lung, there is tympanitic note on percussion and this (131) is most pronounced in the upper lobes where the chest wall is thinnest. (289) If the cavity is large with smooth walls, a metallic tone is added to the tympanitic resonance. If it is covered with thickened lung tissue or pleura, the tone is tympanitic, deadened or absolutely deadened. The same effect is produced if it is filled with secretion, or if the bronchus leading to it is blocked. (132) A peculiar change of percussion note, called Wintrich's sign, is diagnostic of cavity and is as follows. If there is free communication with a/

a large bronchus, the pitch of the percussion sound is higher when the mouth is open, lower when it is shut, and lower still when the nostrils are closed. This sign is found only in connection with tympanicity dependent on pulmonary cavity.(133) In a superficial cavity of moderate or large size freely communicating with a bronchus of medium calibre and having the chest wall thinned by emaciation, percussion yields the crack pot sound, or bruit de pot fêlé. It is best heard in the infra-clavicular region from the 1st to 4th ribs during expiration and when the patient keeps his mouth open.

(134) In very large cavities amphoric resonance is heard; the note is of metallic timbre, higher in pitch and of longer duration that the tympanitic note.

(135) Auscultation over a superficial pulmonary cavity surrounded by rigid and dense walls and large enough to involve one of the larger bronchi so that there is free communication with the trachea, discloses a bronchial breath sound with medium and coarse râles, and resonant râles,(136) though at times these latter lose their resonance.(137) If the cavity is covered with a thick layer of normal lung or the bronchus connected/

connected with the cavity be plugged, this is not
(138)
heard. Where the cavity is very large, as large as the closed fist, an amphoric respiratory murmur
(139)
is heard and the râles associated with it are metallic in character.

(140) The vocal resonance over a cavity under the above conditions is broncho-phonic in character and where amphoric breathing and metallic râles are
(290)
heard, the bronchophony acquires a metallic quality (Laennec's pectoriloquy).

(141) In Pneumothorax, according to Guttman, a tympanitic percussion sound is heard, but if pulmonary fistula be present allowing air to escape, the tension becomes so great that tympanicity is lost.
(291)
Vierordt says that as a rule the sound though very loud and deep is almost always non-tympanitic, but
(292)
sometimes metallic in character. Where the pneumothorax is circumscribed and communicates with the lung by an open fistula percussion may give the
(293)
bruit de pot fêlé and there may be an amphoric breath sound.

TREATMENT OF PHTHISIS.

PREVENTIVE. CURATIVE. PALLIATIVE.

Preventive.

The reduction of the phthisis rate during the last quarter of a century following as it does improvement in the sanitary and hygienic conditions under which the people live, and also the fact that comparatively few who, having once developed the disease even under good conditions, are able to take their place in the routine of life, point to the feasibility and the importance of preventive treatment both applied to the general conditions which favour the spread of the disease and also to the susceptible individual himself. It has been shown that the main sources by which the infection enters the human body are:-

A. By inhalation of particles of dried sputum and dust contaminated by it.

B. By ingestion of tuberculous milk or meat. (Denied by Koch).

A consideration of the predisposing causes of

Phthisis/

phthisis also brought forward prominently, the fact that the development in the body of the infection from the above sources is greatly favoured by overcrowed damp dwellings and workshops associated as these frequently are with deficient and improper feeding. It is by attention to preventing infection from the above sources and also by improving the resisting power of the human body by better surroundings and nourishment, that the main hope of decreasing the amount of phthisis lies.

I. Inhalation of dried sputum or tuberculous dust. This can only be effectively guarded against by diagnosing early cases of phthisis and by as far as possible making every patient destroy his expectoration, and where intestines are affected and diarrhoea is present, by disinfecting the motions. With regard to expectoration, this is best received into a small bottle, e.g., Dettweiler's flash, containing a small quantity of 5% Acid Carbolic solution. This should be emptied down the soil pipe once or twice daily, or else destroyed in a good red fire and the apparatus boiled if possible or washed out with strong solution of soda and after

wards/

wards scalded out with boiling water. In some cases patients object to this and in preference receive expectoration in slips of paper which can be immediately burnt, or if that is impossible, kept in enamelled or glass box which is capable of sterilising by boiling. Flugge has observed that spray of tubercular sputum is very infectious, so that in case of patient with moustache, it would seem advisable not to use a handkerchief to wipe away remains of expectoration from mouth, but rather old rag or piece of paper which can be burnt.

Infection by Milk.

This is the most important source of intestinal affection in infants and young children, who are the largest consumers of milk, and it is worthy of note that while pulmonary phthisis had decreased greatly consumption of bowels has undergone no corresponding modification. The milk can only be infected in the case of a cow with tuberculous disease of the udder, so that not every cow which is shown by the tuberculin test to be tuberculous is able to transmit phthisis by its milk, still it must be a source of future danger, as given the tubercle in one part

of/

of the body, it may in time affect the udder. In order to strike at the root of tubercular infection by milk, it would be necessary to have all the cows used for milking purposes examined by tuberculin and all those who showed the tubercular reaction, either rejected as milkers, or else subjected to a further frequent examination of the udder, and if it is declared free by skilled evidence, the milk might be used after sterlising or boiling for fear of infection through some small unnoticed lesion. If at the same time a higher standard was insisted on in cowhouses both in town and country, if they were better lit and better ventilated, walls and floors partially concreted instead of mere cobble stone flooring, and a minimum cubic space of 600 - 800 cubic feet per animal, as advised by Royal Commissioners on Tuberculosis, (27) it might be possible to stamp out this source of infection. (236) I believe in some sanatoria, etc., tuberculin free herds are kept for the milk supply and in Denmark this has also been done in dairies. A radical reform such as this must be comparatively slow, and it must be a matter of time before one can look for

dairy/

dairy keepers to use only tuberculin tested cows, as that would mean an increase of capital laid out on stock and consequent inability to get a sufficient return by selling milk at the same price as is obtained in the open market, and those who most require to be protected in this way, viz., the working classes, would from inability to pay the price, be excluded from the benefit. Short of this radical reform, it might be possible to have the udders inspected by qualified veterinary surgeons at regular intervals, monthly or weekly and to institute bacteriological examinations of suspicious sores and all cases (with tubercular udders) rejected as milkers. Until something of this kind can be undertaken by municipal authorities, the only safeguard for the public is to have all milk boiled or sterilized by heating in water bath to 180° F., the latter tasting the milk less than the former and the only objection being that both render the milk less digestible and precipitate some of the salts. The supplying of sterilized milk has indeed been taken in hand by the authorities in America and at St. Helens in Lancashire with very satisfactory results in the latter place.

Infection by Meat./

Infection by Meat.

Authorities differ as to the exact amount of danger from this, but it is certainly very much less than from milk because it is never in this country at least eaten in the uncooked state. Moreover it is only very rarely indeed that the fleshy part of the animal, which is used for food, is found affected with tubercle. The tubercular deposit is found almost entirely in the organs, lungs, lymphatic glands, spleen and liver, and serous membranes. Certainly when the disease is active and widespread it must seriously interfere with the nutrition of the animal and deteriorate the fleshy parts and render them less fit for human food, but at the same time, it cannot transmit tubercle except the affected parts be eaten. What is necessary is that the infected parts be removed from the carcase and it is here that danger may arise from a careless butcher infecting sound flesh with a knife contaminated in the process of cutting away infected organs. It is therefore necessary that the greatest care be exercised in this process and different knives be used for cutting away infected organs and the cutting up of fleshy parts, or/

or else the knives should be thoroughly sterilized. Great cleanliness of hands and care in washing after handling infected parts before touching the sound flesh should be taken. It has been found that Tubercle Bacilli in the centre of a joint or roll of meat may escape destruction with ordinary cooking, hence the necessity of very thorough cooking through of the meat so that its whole substance is permeated by a sufficient temperature to destroy the Tubercle Bacilli. The next important point is the attention to hygienic surroundings at home and at work, as a means of improving the organism and by improved ventilation and lighting, hindering the aggressive power of the Tubercle Bacilli. It has been abundantly shown by Ransome and others that dark, damp, ill-ventilated dwellings, more especially back to back houses without any possibility of through draught, such as are found in many English towns, have a great influence in causation of phthisis. This is, of course, complicated by other factors, early and imprudent marriages, found more frequently in the working classes, with their consequent overcrowding and defective nourishment. Much has of late been tried in London and other large/

large towns to remedy this evil in the way of condemning and knocking down old houses and rebuilding them in improved form, but the complaint is made that when rebuilt they are inhabited by a better class, while the old inhabitants drift away and help to further overcrowd other densely populated areas. It seems that the people themselves require educating up to the laws of health and to be taught that transgression of these is sooner rather than later followed by disease. They require to be taught to exercise self control in matters of finery, drink and amusement, and to devote the money thus saved to live in better dwellings and to get better food; and also to be told what food they may get best nutritious value from for their money and how to cook the same well, though plainly. For poor families a great improvement in nutrition and ability to resist disease would follow the employment of oatmeal, peasemeal, lentils, broth, milk, cocoa, cheese, etc., instead of tea, fancy breads, beer, etc. Much is being done by county councils, etc., in the way of cooking classes, but more requires to be done to reach the lower strata of society/

society who suffer chiefly from tubercular disease. With regard to the notification of phthisis, it might be made to help in three ways,

1. If a patient be notified in the early stage he might be given instructions as to the disinfection and destruction of his sputum and also as to the great value of open windows, fresh air, and proper, wholesome food; thus the patient himself would be helped and there would be less risk to his friends. Much, of course, is done by individual members of the profession, but it might be done more thoroughly if it were backed by the authority of the Sanitary or Health powers of a city or district.

2. By this means also, on the termination of a case the thorough disinfection of the room inhabited by the phthisical patient might be seen to by the sanitary authorities.

3. It would also bring under notice houses and groups of houses as centres of infection to successive inhabitants and as predisposing causes by their insanitary condition and they could then be dealt with as seemed necessary.

Individual Prophylaxis.

Heredity has, I think, been proved to transmit a tendency to the disease and members of such families can hold this tendency in check by adopting a certain mode of life. They should live in a thoroughly dry house on a sandy soil on the slope of a hill, and exposed to South and West, and sheltered/

ed from North and East winds. There should be plentiful admission of sunlight and fresh air by means of large windows kept much open. They should have plenty good, plain food with a large allowance of fatty material in the shape of cream, fat bacon, eggs and milk. They should have regularity of meals and an outdoor occupation as far as possible, or at all events, one where they are not worked in stuffy, dark, ill-ventilated rooms.

In cases which seem to thrive badly in spite of this, residence in a dry climate at a high altitude would probably be beneficial, as according to Hirsch (162) this seems to strengthen and develop the respiratory organs and in support of this effect of altitude, he instances the fact that in some high-lying South American towns, e.g., Quito, where insanitary conditions abound, phthisis is absent, or very rare.

Curative Treatment of Phthisis.

How far is phthisis curable? That it is curable in early stages has long been known by the evidence of the Post Mortem table, which showed frequent cases with cicatrices at apex in patient dying/

(257)

dying of the disease. Calwell has, however, criticised this and says though this frequently occurs, it is very rare indeed to find a patient dying of other disease, an apex gone or half an upper lobe cicatrised. In ordinary practice also one rarely comes across cases of phthisis which occurring at an early period of life, have been cured and the patient been able to resume work. In 12 years' practice I can only recall two patients and of these, one has practically lived an invalid life that is, though able to go about, he has never done much work and has lived on his friends almost all his life. There are perhaps half a dozen others who have done well in South Africa, but unable to live in this country. One is then driven to the conclusion that a complete cure is only to be looked for in those cases which come under observation early, and where physical signs are very slight or imperceptible, and this seems to be the evidence even in connection with the open air treatment. Certainly all phthisis patients derive great benefit from it and live longer and have less discomfort but the percentage of absolute cures with marked physical signs even with open air treatment is small/

small. (238) Calwell says, "In consumption, while the majority of cases react well and speedily (to open air), cure appears to be confined to very early and hopeful cases. (239) Of 35 cases under his observation in the Throne Hospital, which gained weight and improved in condition, only ten maintained the improvement for a year or so and one returned to work. The others all fell away during or after the third month and either died in hospital or went home to die. Of the ten, all bore distinct physical signs, only one or two being without râles."

(29) Woodcock of Leeds says, "a sanatorium containing 100 consumptives would, according to German statistics, cure absolutely 10 - 12 patients and almost cure another 20. (30) He also says that Walther of Nordrach told him he had little faith in sanatorium treatment for working classes as they generally have to return to their old surroundings and there is great fear of their relapsing."

(365) Williams says, "If patient is prepared to make certain sacrifices of time, money and liberty for some years, to carry our rigidly certain common sense rules which long experience of the disease inculcate, he may under favourable circumstances live/

live on for a long period, even to the ordinary span of life and as he lives may gain sufficient strength to resume his former occupation and duties."

He gives statistics of 1000 private patients taken from upper and middle classes - all twelve months under treatment.(366) Of these, 20% (198) died; of the remaining 802, in 34 the lungs were healthy; in 280 the lungs were improved.(367) The results with regard to general health were more favourable 35½% being well and able to follow their occupation, and 36½% tolerably well, able to do something, but owing to being subject to relapses, obliged to use precaution. The case for the curability of phthisis may then be put, that if the patient is in the early stage, he may by proper treatment be cured; with marked physical signs, his chance of absolute cure is small, though by attention to treatment, he may live for years, be able to do some work and enjoy comparative health, but will be subject to relapses. This practically shuts out the class which suffers most from phthisis, viz., the working class, as their surroundings are such that if cured in a sanatorium, they will be very liable to relapse in the ordinary competition and worry of everyday life, and/

and if only partially cured, they have little chance of keeping what they have made when they return home. (240) A suggestion has been made by Dr. Jane Walker, that an after care Association be formed for the further care of hopeful cases when they leave a sanatorium. (31) Dr Woodcock suggests the formation of hygienic colonies to allow of transference of cured phthisicals from town to country. This is a most valuable suggestion and seems almost as necessary as sanatorium treatment, and without something of the kind, the undoubted benefits of a sanatorium are largely lost to the working classes.

The cardinal point of curvature treatment is improvement of the surroundings of the patient. If he lives in town, he should be transferred to the country and his nourishment increased; if a country dweller, it will generally be found that his housing is at fault and nourishment defective and these must be at once remedied.

The immediate improvement following the free use of fresh air and full feeding are testified to by all who have any experience in the matter. (33) Dr Burton Fanning says that improvement may confid-

ently/

ently be expected in 90% of cases.(241) The immediate effects which follow the fresh air treatment are, according to Dr Philip of Edinburgh:-

1. Improvement of colour and appearance
2. Increased appetite and assimilated powers.
3. Increase of weight.
4. Lessening of cough and expectoration.
5. Disappearance of night sweat.
6. Improved circulation.
7. Fall of temperature.

In addition to the abundant use of fresh air, the hygienic treatment consists in very full feeding, (though forced feeding to the extent of persevering with eating after vomiting has been produced seems unphysiological, unless vomiting has been caused by cough), regulated rest and exercise and avoidance of all causes of excitement and worry. This treatment can be carried out at home, but is generally more satisfactory if, at all events, begun with two or three months at a sanatorium, as thereby the patient is more thoroughly impressed with the need of the strictest attention to every detail in treatment and/

and he is also isolated from the worries of home and the visits of friends, who are rarely judicious and often do harm in their attempts to help and amuse the sufferer.

Of five cases under my observation that have undergone open air treatment in a sanatorium, all have been much improved in general condition, but none have entirely lost their cough and spit, and the physical signs, though in some cases drier, also have not undergone improvement corresponding to that in general health.

One, an adult male following commercial life, has been able to do partial work - a half day in his office - and has maintained and even increased his improvement, though during the 18 months since he left the sanatorium, he has been subject to attacks of high temperature with increased cough and spit, which have always subsided with a few days in bed. Another, also adult male patient, after nine months at a sanatorium has returned home somewhat improved, but not markedly so. He had, however, while at the sanatorium, a severe attack of haemorrhage followed by spread of disease and acute feverish symptoms lasting for several weeks. He might under/

under ordinary circumstances have sunk under the attack, and I think there is no doubt the sanatorium treatment pulled him out of what seemed a very serious and almost hopeless condition. Previous to going to the sanatorium, he had held his own with open air treatment at home, without being able to shake clear of the disease.

Both the above patients belonged to the middle class and were able to be well attended to at home.

The other three belonged to the working class, but two of them living in the country, had the advantage of being well looked after by the people in whose service they were. The first, a young man of 19 years, developed the disease while working as a ploughman and living in a bothy. He was altogether about five months in two different sanatoria, with an interval of about two months between the two periods. It is now about seven months since he came home from the last sanatorium, and since then, though he has lost flesh a little, he has held his ground otherwise. Pulse quiet, no temperature, appetite good, cough and spit much as they were when he came home; phhysical signs stationary. He has lately gone to an agricultural life in South Africa.

The/

The second Patient, his mother, developed the disease after he had been six months ill. She was also in a sanatorium for about four months and though her spit and physical signs were not benefited much, her general condition was much better and now after several months, though she has lost flesh a little, still holds her own and is able for a little housework.

The third patient, being dependent on charity and the work of a delicate wife, is slowly losing ground 18 months after his return from the sanatorium. He was much improved in health and strength by his stay, but has not done any work beyond going round with a pack as he feels able.

In one case, treated at home, with well marked physical signs, fever and night sweats and showing no improvement after a few weeks treatment with open windows indoors, after she had been carried right out into the open air for several hours daily, a very marked improvement set in. The physical signs got drier, cough and spit almost stopped, fever disappeared and she put on flesh and got strong and able to go about again. She kept her ground through the summer, but in the late autumn she/

she had a return of fever, cough and spit. The attack subsided, but was followed shortly after by an attack of influenza which was epidemic at the time. After this, she gradually went down and died exhausted with albuminuria and diarrhoea in a few weeks.

In another case which had an acute onset, the progress of the disease has been greatly checked by open air treatment though the patient has never been able to work. The disease, however, is not arrested and he slowly loses ground.

Sanatoria.

(33) Ransome gives the condition to be filled by sanatoria as laid down by Leon Petit after Turban.

1. Surrounding air must be pure, hence it must not be near a large town or factory.

2. The soil should be sandy without humidity, so as to give the smallest amount of mist or fog after sunset. Plains well exposed to South or broad valleys fully open to the sun are practically favourable, provided they are sheltered from North or North East winds. Pines are valuable both as shelters from heat and wind and purifiers of air.

3. An extensive view is beneficial for its mental and moral effect - an elevated site and gentle slope indicated - good water supply necessary, preferably spring.

4. Easy of access to railway station.

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(34) A description of some American and German Sanatoria in British Medical Journal of April and (198) May shows that they all made it a strong point to have shelter from North and North East winds and are all built on a slope at various elevations varying from about 700 feet at Hohen Honnef up to about 2000 at the Adirondachs. The bedrooms are all made to face South and outside shelters provided either in spots specially sheltered by trees and position from cold winds, or as in Adirondachs, screens of glass etc., are provided. In some, the propinquity of pine woods is thought to bear additional advantage. The treatment in all consists of keeping the patient in an open air atmosphere during the whole 24 hours, those with high temperatures and pneumonic complications being kept in the recumbent posture either in their own beds near an open window as at Nordarch (35), or as at Adirondachs (36), resting in open air outside shelters. (242) At Falkenstein patients with commencing fever are kept in bed, but chronic febrile cases which do not subside with this, are found frequently to lose their temperature with uninterrupted rest in the open air.

After/

After the temperature has become normal, regulated exercise is allowed the patient on easy gradients, the effect on Temperature and pulse rate being carefully watched. If there is a rise of temperature or much increase of pulse rate, the exercise is given up for a time, and if no unfavourable results follow, it is gradually increased. The element which has to be guarded against is wind, which seems to have a prejudicial effect if patient is much exposed to it, probably due to the extra exertion in walking against it and to the chilling effect on a patient exposed to its full force.(243) Dr. Jane Walker, Norfolk, takes her patients out in all weathers except in high wind. At Nordrach, it is found that no amount of exposure to weather or variation of temperatures, cause patient to take cold and the chief attention is directed towards avoiding over exertion.(37) Dr. Gordon of Exeter from observation of several rural sanitary districts of Devonshire, found that those exposed to West and South West winds suffered more from phthisis than the sheltered ones, and that soil only appeared to exercise a subordinate influence/

fluence.[38] Sir W. Broadbent said that windiness and not any particular wind was the injurious agent and acted by causing people to shut themselves up indoors.

An equally important point is attention to the feeding of the patient. In Nordrach three meals a day, and in Falkenstein four meals are given. In Hohen Honnef and Goebersdorf, five meals a day, but in whatever manner the meals are divided, the principle is to get the patient to take as much nourishment as he can possibly assimilate.

[39] At Nordrach the meals are as follows:-

Breakfast at 8.30 a.m., consists of coffee, bread and butter and cold meat, such as ham, tongue, sausage, etc.

Dinner Two hot courses of meat or fish and meat served by the doctor to each patient with an abundance of potatoes and green vegetables and sauces in which butter is a main ingredient. A third course is given of pastry, farinaceous pudding, fruit or ice cream with coffee.

Supper is usually one hot course as at dinner and one cold as at breakfast with tea.

At each meal, the patient has half a litre of milk, as soon as possible reduced to a quarter litre or stopped, according to his capacity or need of putting/

ting on flesh. At the two latter courses, the plates may not be taken away until all has been eaten or the doctor has given permission, and if patient is sick before or during a meal, he comes back and finishes, usually finding no difficulty. The long interval between meals aids complete assimilation.

(40) At Hohenhonnef, one litre of milk is given at breakfast, another litre in the middle of the day between meals and another in the evening. It is, however, stopped if it causes dyspepsia.
(244)
Dr Karl Hess of Falkenstein says that, "being quite convinced of the extraordinary dietetic value of milk, we always cautiously administer it, even where patient states positively that they cannot take it, and suggests addition of one third acorn coffee, or cognac three-fifths of a teaspoonful to a quarter litre or lime water in these cases, and also emphasises the fact that it should be drunk slowly and in small draughts."

Alcohol. is allowed in some sanatoria.(41) In the Adirondachs, none is allowed in early cases, but a small amount is given in acute cases. At Hohen-

honnef/

honnef, a bottle of Rhenish wine is allowed at lunch and dinner; no spirits. At Nordrach, as a drug or beverage, it is strictly limited.(245) At Falkenstein, alcohol in the form of wine or beer is allowed, regulated by doctor to ordinary cases accustomed to its use. Strong doses are only given temporarily in cases of severe illness, or collapse in the form of cognac, champagne, or strong wine. A small glass of cognac in a glass of milk just before going to sleep is recommended to prevent night perspiration. It is contraindicated in haemorrhage unless heart weakness should require it. Rest before and after meals in patients who are taking a considerable amount of exercise is important. It should be for about an hour before and an hour to an hour and a half after meals.

(42) At Goebersdorf hydrotherapeutics are used in the form of a tepid douche after walking and a cool pack for half an hour.(246) At Falkenstein the patient is rubbed down with a dry towel every morning and in stronger patients, water or spirit and water sponging precedes the rub down. In cases of/

of slight disease, a short, sharp douche of ten to thirty seconds is given, followed by a good rubbing down and a short walk.

Drug treatment at Sanatoria is only used as required to meet special symptoms and as little as possible. With regard to drugs as a curative element in phthisis, (247) Dr Caldwell in Throne Hospital, Belfast, tried for successive periods the use of Iron and tonics, Petroleum, Creosote, Gaiacol and Ol. Morrhuse, without observing any effect on the course of disease. (206) Koch tried administering Arsenic and carbolic acid in as large quantities as possible in guinea pigs previously inoculated with tuberculosos, but found no alteration in the course of the disease. (207) He also tried the inhalation of volatile, gaseous, germicidal substances in the case of inoculated mice and guinea pigs, and found that some of the mice died of pneumonia, but in all the others the disease ran its usual course without being in any way influenced by the inhalation. (261) Ransome has tried inhalations of Oxygen, Ozone, and Ozonised Oxygen. Oxygen was found to produce no definite effect of the progress of disease.

Ozone/

Ozone allowed to pass from a generator into wards gave no satisfactory results. Ozonised Oxygen inhaled directly from a Tobold's Gasometer was found to have a favourable effect while the patient was under treatment, although many of them developed active disease after their return home. The fever and night sweats decreased, appetite improved; general gain in weight and strength and colour of blood followed. In my own experience, I have tried Creosote, Guiacol, Iodoform, and laryngeal injection of menthol without any effect on the course of the disease. The Iodoform appeared to do more harm than good by deranging the patient's stomach, while Creosote in small doses appeared to have no special effect on the course of the disease and even when given in doses of 40 m + d. no definite effect was observed and it had to be stopped as it seemed also to interfere with patient's appetite. Guiacol I tried in one case of acute phthisis, by hypodermic injection increasing the dose gradually till mX. night and morning were given without any effect on the course of the disease. Cod Liver Oil is very highly spoken of by Williams/

Williams, but it should rather be looked on as a food than a drug and is certainly not a specific though a good adjuvant. Koch's Tuberculin was tried and found wanting and in the second preparation of Tuberculin, it has by no means proved a specific. Drugs certainly have an important place in relieving symptoms, but as a curative element, the open air method is the only one worthy of consideration.

Residence Abroad.

In early cases with slightly marked physical signs, little or no febrile reaction, there is no doubt a change to a more sunny climate and open air life often arrests the disease, but at the same time, it has often been fraught with distress and disaster to patients in whom the disease had got a firm hold. The patients who go abroad may be divided into two classes. Those who have plenty of means and in whom the disease is more or less advanced, but at least not very active at the time, but by virtue of their money, which does to a great extent procure them home comforts, are justified in going to seek a more genial winter resort in the/

the Riviera, Maderia, Davos, etc., and by such means, there is no doubt the disease is kept in check for years; but there is another class who can with difficulty raise money to take them abroad and who go chiefly with the idea that after a few months' residence, they may be able to make a livelihood in the land of their adoption. These latter cases must be very carefully chosen, as though there is no doubt that cases in an early stage are able after a few months' residence in the high lying plateaux of South Africa and California to earn their livelihood by some outdoor occupation. At the same time, I have never seen any good arise from patients in whom the disease has lasted some time, and associated with febrile reaction going to South Africa. Their best chance would seem to lie in sanatorium treatment at home, and possibly emigration afterwards if the improvement seemed to justify it. As the disease undoubtedly seems less likely to recrudesce in the high altitudes of South Africa and California with their greater sunshine, than it does in this country. (368) It has been found that mountain climates with their stimulating effect on the vital processes and the expansion of thorax/

thorax which they cause (369) are specially suitable as a prophylactic in cases with strong hereditary predisposition in whom the disease is threatened and in cases of imperfect pulmonary development, (370) and in cases of chronic pneumonia or pleurisy where the lung does not expand after absorption of the fluid. (371) As a curative agent they are chiefly useful in phthisis with pneumonic consolidation, or chronic tubercular phthisis where the lung is not too much involved to admit of lung expansion and in first stage of phthisis with slight physical signs and symptoms of only a few months' duration. While it is found that in emphysema, chronic bronchitis, cardiac and kidney cases or those in which large areas of lung are involved, or where the nervous system is irritable, the mountain climates are harmful. (79) Cardiac cases, where there is only a damaged mitral valve with good compensation, may, however, be sent. (80) To get the full benefit, it is necessary that the patient should reside continually in it for two winters and one summer. Those who come to England for the summer should not do so before May and should descend gradually from the/

(372)
the high altitude stopping at intermediate places.
(81)
For cases with large secreting cavities, or in phthisis with bronchiectasis or emphysema, dry climates, such as Egypt and the Cape are found very suitable, as the secretion becomes lessened and the patient breathes easier.(82) Cases with albuminuria, where lung destruction is not too great, also those with insomnia and initable nervous system also do well, but laryngeal and intestinal complications are aggravated.

Marine Climates, e.g., Mediterranean Coast,(373) are specially indicated in cases of phthisis with pneumonic, broncho-pneumonic or pleuro-pneumonic complications or originated by them.(82) Laryngeal cases sometimes improve and coast climates generally(83) are invaluable for children of scrofulous tendency.(374) Chronic phthisical cases sometimes do well in the Riviera; cavities contract, consolidation becomes fibroid, or if case too far gone, the status(375) may long be maintained. Elderly people with bronchitic asthma or emphysema also do well in this climate. Patients should not go to Riviera before November, thereby escaping diarrhoea and/

and mosquitoes and should not return to England before beginning of June.(84) For catarrhal phthisis chronic bronchitis, laryngeal catarrh, and emphysema with scanty expectoration, the moist climate of Madeira is eminently suited;(376) the cough becomes softer, expectoration freer and whole respiratory tract soothed by the mild atmosphere.(85) For vigorous patients with good vitality, the East coast health resorts of Britain are suitable during summer, while for the less vigorous, markedly catarrhal, or febrile cases, the South West Coast is best. (377)

For Sea Voyages the most suitable cases are haemorrhagic, scrofulous with fistula, cases with bad cough and little pyrexia or consolidation, emphysema and overworked cases.(86) Ocean voyages are sedative to nervous system; appetite and digestion are improved. A long voyage, such as that to Australia is necessary to get full benefit of the climate and of course, proper feeding and ventilation of cabin should be assured.

Palliative Treatment of Phthisis.

In those cases which seem not to be able to resist the inroads of the Tubercle Bacillus, there is/

is often considerable relief to be given by treating the troublesome symptoms as they arise.

Fever. This is one of the prominent symptoms. Frequently general means, rest in bed, fresh air, are sufficient to keep it under control, or if these are insufficient, cold packs or sponging are sometimes effectual. Iced packs have been used with good results in acute phthisis with very high Temperature, but have to be carefully watched and remitted on any appearance of collapse impending. Quinine and the Antipyretics of the Coal Tar group, if given in sufficient doses, will reduce temperature, but their effect is only temporary and are apt to produce collapse. I have found the combination of Quinine and Phenacitin given three or four hours before the onset of the afternoon rise of temperature effectual in some cases in preventing the rise and making the patient more comfortable. In continued moderate pyrexia of early stage, Quinine and Arsenics are said to be beneficial, though personally, I have found little benefit from their use. From my own experience, I should be inclined to place rest and fresh/

fresh air first and in cases resisting these, try repeated cold sponging or Quinine and Phenacetin in addition, but only as adjuncts to the general means.

With regard to treatment by Antistreptococcus serum, which would be suggested by the fact that probably Streptococcus injection is accountable to some extent for the fever. (262) Ransome has recorded two cases in which it was used. In the first case, one of acute phthisis of about six months duration with severe fever and marked streptococcus chains in the sputum, little, if any good effect followed three injections of antistreptococci serum at 12 hour intervals, and on the injections being repeated later on, they occasioned so much distress, that they were given up. In the second case, which began apparently as a septic pneumonia with irregular high temperature, becoming after a couple of months less acute and temperature more regular, but always showing evidence of rise and now showing Tubercle Bacilli in sputum and also a pure cultivation of Streptococcus in glycerine agar, injections twice a day of 10 cc. and then 20 cc. were given; after the fourth day injection was only made once/

once; on the sixth day an irritating erythematous rash came out. After beginning the injection, the Temperature slowly but steadily fell, until on the seventh day it became normal in the morning, but rose to 103.4 in the afternoon. However, after stopping the injection, it immediately rose again, showing that no permanent benefit had been obtained.

Cough. The patient must be taught to resist the inclination to cough as much as possible and to limit the cough to the bringing up of expectoration. Under fresh air treatment, it generally becomes quickly reduced to a minimum. If the cough resists these means, a stimulating expectorant such as Ammon. Corb. gr. with a few drops ( ) of Liq. Morph. Hydrochlor. is often useful. Medicated inhalations of Ol. Eucalypt., Acid Carbol., Chloroform or Creosote given with a steam inhaler or atomised with an Oppenheimer atomiser from a solution in Parolein. If cough very severe and spasmodic, I have seen great relief follow the intra-laryngeal injection of 10 - 20% Menthol and Ol Oliv. solution

being/

being used at times, but it is frequently necessary to fall back on opium, which I have generally given in the form of Pil Opii, ½ - 1gr. being given at bed-time.

Pain.

To relieve attacks of pleuritic pain, strapping the affected side with plaster, or the application of a rising blister or warm poultice generally give relief. If very severe, a small hypodermic of Morphia $\frac{1}{8}$ with $\frac{1}{200}$ atropine to $\frac{1}{6}$ with $\frac{1}{18}$ of Atropine may be given.

Anorexia and Sickness.

An alkaline mixture of Soda and Rhubarb with a bitter infusion, or Begbie's mixture often helps; if vomiting considerable, restriction to peptonised milk, raw beef juice, or white of egg, whipped up with a little sherry or brandy is often more effectual than drug treatment.

Night Sweats.

Avoid over wrapping patient and over heating room: sponging with vinegar. Have hair cut short. Pil Atrop. Sulph. gr $\frac{1}{75}$ - $\frac{1}{150}$, I have found the most effectual/

effectual means of checking sweat. Camphoric acid and Picrotoxin are also highly recommended, but since using open air treatment freely, I have seen very little necessity for special drug treatment.

Diarrhoea.

In early stages best treated with a dose of castor oil followed by a special diet of boiled milk, scraped meat, arrowroot, and if this is insufficient Bismuth Sub. Nit. or P. opii gr. ½

In the later stages of the disease where it is due to waxy disease and ulceration, a pill of Cupri Sulph. gr. ¼ - ½ Ext. opii gr. ½, Ext. Haemotox. q.s. is very efficacious, or a mixture of Acid Carbol gr. $\frac{\cdot}{I}$ Acid Tannic gr. $\frac{\cdot\cdot}{II}$ -V generally keep it pretty well in check.

Ulceration of Throat and Mouth.

In early stages Mellis Boracis, or a Pot. Chlor. and Myrrh gargle. If much dysphagia, a 4 - 10% cocaine spray before meals or insufflation of Morph. Hydrochlor. gr. ¼ Bismuth gr. XV. will relieve considerably. It has also been recommended for patient to swallow his food while lying on his face.

Haemoptysis.

For slight haemoptysis of early stages, rest in bed is generally sufficient. For more severe cases many drugs have been recommended as to the utility of which doubts have been freely expressed. Ergot, Hydrastis, Homomcis Digitalis Gallic and Tannic Acids, Lead. I have certainly seen severe haemorrhage arrested by Tannic & Gallic Acids with a small dose of morphia added, and am inclined to think they are sometimes beneficial. Nauseating doses of Ipecacuana are strongly recommended by Mousseau and would probably be beneficial by lowering the blood pressure, but at the same time, the risk of incurring further haemorrhage by the onset of vomiting seems to be considerable. Calcium Chloride, by its power of increasing the coagulability of the blood, would seem theoretically to be beneficial, but it failed completely in a severe case in which I tried it, and which was arrested by a mixture of Ergot, Tinct. Digitalis Tinct. Opii.

Opium in one form or another seems to be the only drug that is umiversally spoken well of. It, at/

at all events, soothes the patient and allays nervous anxiety and restlessness. Common salt is a domestic remedy and probably acts by its nauseating properties and its effect on the blood perhaps increasing its coagubility. In severe cases Assalinis bandages by keeping blood in extremities are useful. Ice bags to chest, I have tried also, both constantly and intermittently without seeing much benefit result.

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